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Attention-based maintenance in the working memory of children and adolescents with attention-deficit/hyperactivity disorder

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Abstract

Working memory is the cognitive system dedicated to the temporary storage and processing of information to accomplish goal-directed behaviour. Its functioning is closely intertwined with the functioning of attention, a cognitive resource used by typically developing individuals to boost memory representations held in working memory and maintain information therein. Rare studies in the literature investigated the use of attention-based maintenance in working memory by clinical populations suffering from working memory alterations, such as children and adolescents with attention-deficit/hyperactivity disorder (ADHD). The clinical literature has for long reported that ADHD is characterized by impairments in working memory functioning, yet mechanistic accounts of these deficits lack in the literature. In this thesis, we propose an experimental approach to identify the locus of the working memory deficits in ADHD. Through a series of three experiments, we investigated if children and adolescents (10 to 16 years) diagnosed with this disorder can orient attention and use it to refresh items during classical working memory tasks. Our results showed evidence suggesting that these two mechanisms of working memory functioning are intact in this population. We discuss our results according to the theoretical frameworks of the time-based resource-sharing model of working memory (TBRS) (Barrouillet et al., 2004; Barrouillet & Camos, 2015, 2021), and the working memory model of ADHD (Rapport, Chung, et al., 2001; Rapport, Kofler et al., 2008; Alderson et al., 2010).

To all undergraduate students with an interest in experimental psychology, for I was once them in embryo.

For the same reason, to my parents.

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Introduction

Psychological science has always been interested in describing the human mind and behaviour, and the interplay between them that engenders human experience. Of utmost importance is the mind's capability of briefly suspending the course of action to link past, present, and future events. This permits the execution of goal-directed behaviours and leads to optimal interactions with the surrounding environment. This ability requires a cognitive apparatus that allows one to somehow process incoming information and respond to it properly. Without it, humans would be nothing but automata reacting to stimuli when prompted. This powerful cognitive apparatus – working memory – is the topic under investigation in this doctoral thesis.

Working memory is the human ability to mentally hold, process, update and transform information during short-term delays in order to accomplish complex mental tasks. It is used in all everyday life activities involving one's conscious engagement with the environment, from reading the newspaper, having a conversation, to driving a car or baking a cake. As such, working memory can be undoubtedly considered the keystone of human cognition and it is therefore implied in many neurodevelopmental disorders affecting cognitive performance.

Working memory's central role in human cognition makes it one of the most important research topics in cognitive psychology since the very beginnings of the discipline in the 1960s, when the term was coined (Miller et al., 1960; Atkinson & Shiffrin, 1968). Since then, different definitions and theoretical models have been proposed to explain working memory's functioning, structure, and capacity limits, most of them built upon evidence from experimental and neuropsychological studies with healthy and clinical populations. This thesis addresses theoretical issues on working memory modelling by translating the predictions of the Time-Based Resource-Sharing model (Barrouillet & Camos, 2021) to a clinical population known for their deficits in working memory functioning: the children and adolescents with attention-deficit/hyperactivity disorder.

The attention-deficit/hyperactivity disorder (ADHD) is the most frequent neurodevelopmental disorder in childhood, affecting an estimate of 5% of children (Polanczyck et al, 2007). The psychiatric literature has for long reported that working memory functioning is altered in this population (Kasper et al., 2012), but mechanistic explanations in terms of *which* working memory processe(s) is(are) affected in ADHD are lacking in the literature. Here, we propose that the Time-Based Resource-Sharing model offers a theoretical framework able to examine and maybe account for the weaknesses in the working memory performance of these individuals. Working memory functioning and attention-deficit/hyperactivity disorder are far from being unrelated topics. Yet, basic experimental research on the former and applied clinical research on the latter seldom encounter each other in the scientific literature. This doctoral research is an endeavour to bring these two research fields together – or, at least, a bit closer.

In Chapter 1, we will introduce three contemporary models of working memory and present data on how this cognitive system develops across the lifespan in typically developing individuals. In Chapter 2, we will expose the symptomatology, epidemiology, and aetiology of the attentional deficit/hyperactivity disorder, and then summarize three theoretical models of this disorder that focus on basic cognitive processes, including working memory. From Chapter 3 onwards, we will present three experimental studies aiming at locating the source of working memory deficits in children and adolescents with ADHD. In Experiment 1, we tested if children with ADHD can orient attention to stimuli at encoding and to mental representations during maintenance by using the cued recognition task. In Experiments 2 and 3, we tested if children with ADHD can use attention to refresh items held in working memory by using the complex span paradigm. By the end of this thesis, we will draw a comprehensive framework integrating these two research fields, and, ultimately, show that experimental research on individuals with attention-deficit/hyperactivity disorder can serve as a powerful tool to develop research on working memory.

Part I

Theoretical framework

Chapter 1

The development of Working Memory

The ability to actively hold information in mind and use it to carry out tasks is ubiquitous in day-to-day activity. From holding a conversation with a stranger to driving home safely, working memory ensure us that we will not forget the name of the person in front of us nor cross a red light when our favourite song starts to play on the radio. All that thanks to its faculties of maintaining, processing, and updating information on-line. In this chapter, we will present three theoretical models of working memory and explain their similarities and differences. Then, we will address the developmental changes in working memory across the lifespan, with a focus on how its inner mechanisms are optimized to maintain information.

1.1. Working Memory: Definition and models

The term "working memory" is used in the literature to refer to different conceptualizations and theoretical frameworks (see Cowan, 2017, for a complete discussion on the topic). Here, we adopt the definition of working memory as being the ability to temporarily maintain and process information applied to an ongoing task. All the theoretical models that we shall refer to in this thesis are consonant with this definition, but they diverge in regard to specific components of working memory, and their functioning.

The contemporary models of working memory can be divided in two broader groups, each of them describing its architectural features, properties, and functional processes therein. The first group can be characterized as "modular models", as they focus on the description of interdependent structural modules dealing with information of different sensorial modalities. The focus of their explanation is, therefore, on the nature of the material held in working memory and the specific mechanisms employed by the system to maintain it. The second group of models is formed by "non-modular" models that share

the basic assumption that memory is a non-compartmented cognitive system in which information can take different levels of activation. These models therefore focus on describing processes and mechanisms rather than the architectural components of human memory.

1.1.1. The multicomponent model of working memory

The classical instance of modular models is the multicomponent model of working memory, initially proposed by Baddeley and Hitch (1974) and later developed by Baddeley (1986; Baddeley et al., 2011) and Logie (1995; Baddeley & Logie, 1999). The authors conceptualize working memory as "a limited capacity system for the temporary maintenance and processing of information in the support of cognition and action" (Baddeley et al., 2021, p. 10). We draw the reader's attention to the fact that the authors use the word "system" in their definition, illustrating their view that working memory is a set of separate - but functionally interconnected – components (modules).

In the early formulations of the multicomponent model, working memory was conceived as a tripartite system encompassing the central executive component and two subsidiary components, specific to sensory modalities: the phonological loop and the visuo-spatial sketchpad. The central executive is a capacity limited control structure of working memory. The authors intentionally avoided to address the complexity of the central executive and used it as a "homunculus-like" placeholder to describe a set of executive processes in working memory (Baddeley, 1996; Baddeley & Hitch, 1974; Baddeley & Logie, 1999; Baddeley et al., 2021), for instance, attentional allocation and coordination of the subsidiary systems. The two subsidiary systems, in their turn, are responsible for the processing and the temporary maintenance of information from a particular domain and/or sensory modality. The phonological loop is specialized in language-related and acoustic information (i.e., speech, lipreading, sign language, music and sounds) (Rönnberg et al., 2004), whereas the visuo-spatial sketchpad is specialized in visual (color, shapes), spatial (locations and movement sequences), and haptic (kinesthetic and tactile) information (Baddeley et al., 2011).

Each slave system possesses its own inner mechanisms to prevent information from decay. The phonological loop consists of a phonological store and an articulatory loop that reactivates decayed memory traces via phonological rehearsal, that is, the subvocal repetition of verbal content. This rehearsal process maps onto brain areas involved with

motor planning for speech production and perception (Baddeley et al., 1975; Baddeley et al., 1984). In its turn, the visuo-spatial sketchpad is further subdivided into a passive visual cache that holds purely visual information (i.e. static visual patterns and object's visual features like color, shape, texture) and an inner scribe able to actively rehearse dynamic spatial information (i.e. spatial sequences) (Baddeley & Logie, 1999; Logie & Marchetti, 1991; Logie & Pearson, 1997). Analogously to the phonological loop, which relates to verbal-acoustic perception and production the visuo-spatial sketchpad relates to visual perception and visual imagery, but the exact nature of these relations is less clear (Baddeley & Andrade, 2000; Borst et al., 2012).

This fragmentary view of working memory was proposed by Baddeley and collaborators in opposition to the unitary short-term memory store (STM) in the model by Atkinson and Shiffrin's (1968, 1971), who did not specify further subdivisions in STM based on control processes nor in the sensory modalities of the material – albeit they acknowledged this possibility. One should note that the idea of modality-specific systems in human mind was not novel to cognitive psychology at that time (see, for instance, Brooks, 1967), but Baddeley and collaborators were the first to apply it to the study of working memory in a straightforward manner. Through series of dual-task experiments (Baddeley & Hitch, 1974; Hitch & Baddeley, 1976; Logie et al., 1990), they demonstrated that performance in reasoning, memory for verbal material and for visuospatial material were better explained if one assumed that they are carried out by separate modules in working memory (the central executive, the phonological loop, and the visuo-spatial sketchpad, respectively).

Later, Baddeley (2000) added a fourth component to the multicomponent model in order to account for multimodal binding of information and data not explained by the original proposal. For instance, blocking specific rehearsal strategies and/or exceeding the capacities of the phonological and visuospatial loops does not yield a complete drop on performance, which suggests that this information is stored elsewhere. Baddeley uses the example of memory for unrelated vs. related words: in the former case, participants usually cannot recall more than 3-4 words (i.e., the limit of the phonological loop); but in the latter they can recall up to 16 words if they form a meaningful sentence (i.e., the items are stored in chunks of semantic information). The new component accounting for these phenomena was named "episodic buffer" as it is responsible for creating and maintaining unitary representations by integrating information from the two slave systems, long-term memory,

and possibly form smell and taste. The episodic buffer was originally conceived as a purely passive store, in a sense that it does not possess its specific rehearsal mechanisms as the phonological loop and the visuo-spatial sketchpad. However, in the last conceptualization of the model (Baddeley et al., 2021), the authors acknowledge the existence of an attention-based rehearsal mechanism possibly acting on memory traces in the episodic buffer. Figure 1 shows the conceptualization of the multicomponent model after the inclusion of the episodic buffer.

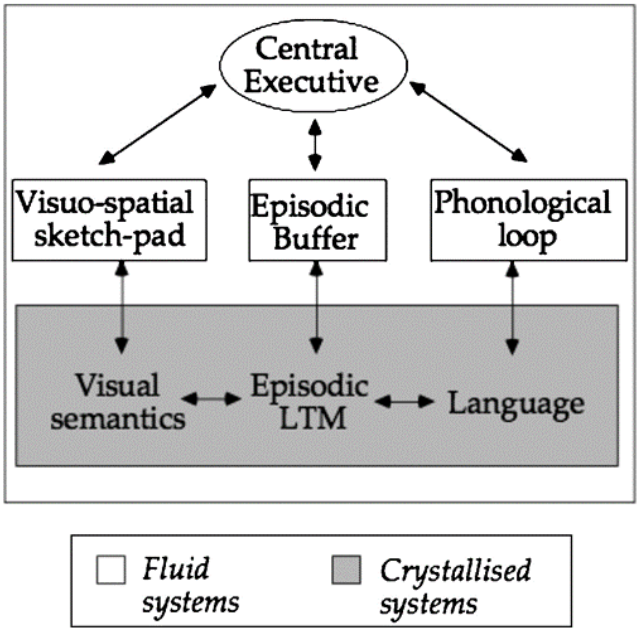

Figure 1. The multicomponent model of working memory, in the version proposed by Baddeley in 2000. Image reproduced with authorization (Baddeley et al., 2011).

Lastly, long-term-memory does not require any controlled process or mediation from the central executive component to access all the three subsidiary systems in the multicomponent model. It has direct access to them via language (i.e., speech, access to the phonological loop), visual semantics (i.e., written signs and symbols, access to the visuo-spatial-sketchpad), and episodic long-term representations (access to the episodic buffer). Conversely, information can be retrieved from long-term memory via mediation from the slave systems and the control of the central executive. For instance: when one

tries to remember the capital of Brazil, the central executive coordinates a "search" in semantic memory that might activate phonological representations ("it starts with a B, with a Bra…Brasília!") and episodic long-term representations (geography lessons from second grade; getting lost in the city during a scientific congress), and some components of the visuospatial sketchpad ("the city's urban plan looks like a plane!").

In short, the multicomponent model of working memory posits a central executive module that coordinates attentional allocation and the conjoint action of the three slave subsystems, and the direct access of both sensory information and long-term memory knowledge to the subsidiary modules.

1.1.2. The time-based resource-sharing model of working memory

A second instance of modular theories of working memory is the time-based resource-sharing (TBRS) model by Barrouillet et al. (2004; Barrouillet & Camos, 2015, 2021). The authors conceptualize working memory as "the structure in which mental representations are built, maintained, and modified according to our goals." (Barrouillet & Camos, 2021, p. 86). Here again one can note the emphasis given to the architectural description of working memory – it is a cognitive *structure* maintaining information and performing mental computations in service of goal-directed behavior.

The core postulate of TBRS model is that limited attentional resources must be shared between the maintenance and processing of information over the time course of a mental task. This implies a constant and sequential attentional switching between these two processes, that is constrained by the cognitive load of the task (Barrouillet & Camos, 2012). The model conceptualizes cognitive load as the ratio between the time that a given task captures attention – thus precluding other attention-demanding processes - and the vacant time to perform it (Barrouillet et al., 2007; 2011). The more attention is available to be engaged in maintenance (e.g., after being disengaged from a concurrent processing task), the lighter is the cognitive load. In other words, with free time, one can use attention to refresh memory traces and avoid temporal decay. Conversely, if a task does not allow one to divert attention from processing and focus it on the memoranda, the memory traces cannot be refreshed and will suffer from temporal decay at a higher rate. Note that the authors' definition of cognitive load is not intrinsically dependent on the task's nature: for instance, a simple task like reading letters can be more detrimental to working memory

performance than reading full sentences if the pace of the former occupies attention uninterruptedly (Lépine et al., 2005).

In terms of structure, the TBRS model is similar to the multicomponent model in a sense that it posits a central module that interacts with long-term memory and with peripheral systems devoted to the maintenance of modality-specific material (Figure 2).

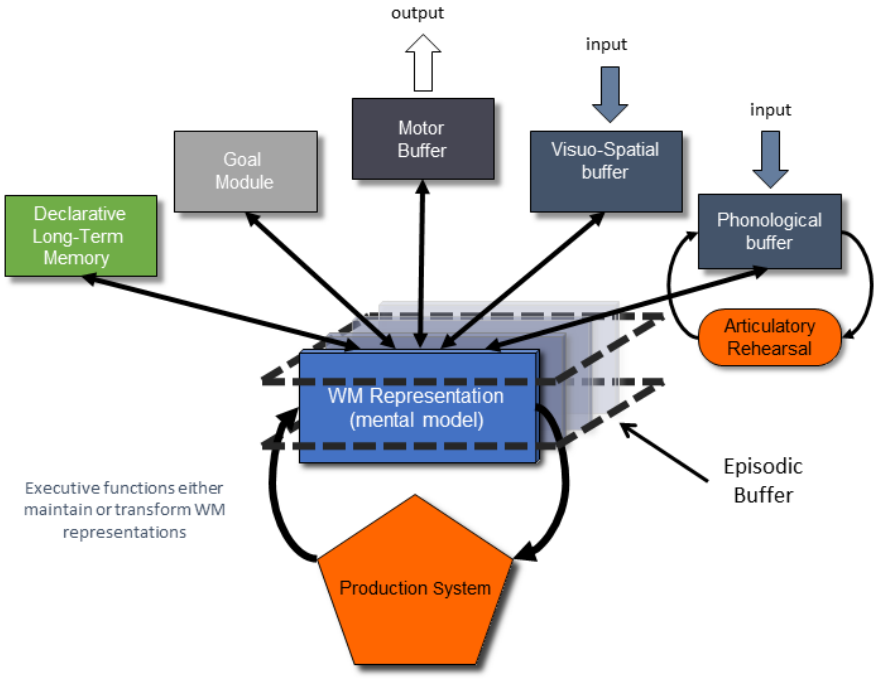

Figure 2. Time-based resource-sharing model of working memory. Image reproduced with authorization from Barrouillet and Camos (2005).

The central module in the TBRS consists of an episodic buffer where mental representations are built and maintained. These mental representations are formed by information coming from the peripheral subsystems (e.g., verbal information from the phonological buffer and declarative long-term memory) and are continuously read, maintained, or transformed according to procedural rules of a production system. This production system is based on long-term knowledge and consists of internalized rules such as "*if…then…*" structures that allow mental operations on the content of the episodic buffer. In the TBRS model, the content of the episodic buffer is under the focus of attention, which implies that "to pay attention to something" means to consciously form and manipulate representations in the buffer. The continuous scanning and transformation of

those representations by a procedural system is called the executive loop in the model. One should note, however, that the executive loop in the TBRS model does not act as a central controller of the system nor is entitled to homunculus-like properties. Rather, the authors defend that cognitive control is an emergent property of working memory during goal-directed behaviour – thus not requiring an executive module as in the multicomponent model (Barrouillet & Camos, 2021; see also Vandierendonck, 2016, 2021 for another non-centralized account of executive control in working memory).

The peripheral systems in the TBRS model provide elements for the construction of mental representations by the executive loop. The model posits the following peripheral systems: the phonological buffer, the visuospatial buffer, the motor buffer, an articulatory loop, episodic long-term memory, and declarative long-term memory. The former two subsystems (the phonological and visuospatial buffers) are modules that receive sensory information from the auditory and visuospatial domain, and then forward it to the episodic buffer to supply the executive loop with material. The motor buffer is a module that receives output from the executive loop and execute motor responses (e.g., a key press). The word "buffer" in the components' names emphasize the transient nature of the information therein: they serve as gateways and waiting rooms for material (i.e., sensory information, motor responses) entering and leaving the executive loop.

The articulatory loop is responsible for the articulatory rehearsal of verbal material by means of motor programmes involved in speech production. It should be noted that, in the most recent conceptualization of the TBRS (Barrouillet & Camos, 2021), the articulatory loop is conceived as a purely articulatory system, not coupled with a phonological store that buffers symbolic representations – as in the multicomponent model (Baddeley, 2000). This means that the articulatory loop does not store phonological representations *per se*, but rather uses speech programmes to reactivate verbal content not necessarily held in a specific store. This idea was put forward by Barrouillet and Camos (2021) based on results from the maxispan procedure (Barrouillet et al., 2021). In the maxispan task, the articulatory capacity of the loop is overloaded, and participants are instructed to use attentional-based rehearsal (named attentional refreshing, rather than articulatory rehearsal) to maintain the exceeding items. Not only this procedure dramatically increased the mean span in about 1.4 items compared to the simple span procedure, but it also made the phonological similarity effect (i.e., the higher recall performance for phonologically dissimilar compared to similar items) disappear. After these data, the authors suggest that the items maintained through articulatory rehearsal

might be stored under a non-phonological code (otherwise the similarity effect would have mandatorily manifested), and that the articulatory loop not necessarily contains a phonological store.

Finally, declarative long-term memory supplies the executive loop with semantic information from one's knowledge basis and episodic long-term memory provide information about the task's goals. Both declarative and episodic long-term memory have bidirectional links with the executive loop, meaning that their content can be altered by operations of the executive loop. For example, while performing mental arithmetic calculations, the mental representations in the episodic buffer can leave traces in declarative long-term memories in the form of mathematical facts. Similarly, the subgoals of a mental computation are updated during its execution, according to the results of the operations in the executive loop.

The TBRS model postulates two dissociated maintenance mechanisms in working memory (Camos et al., 2009; Mora & Camos, 2015): articulatory rehearsal and attentional refreshing. They can be employed conjointly to avoid decay of both verbal and visuospatial information, and adults are able to segregate them to optimize maintenance (Barrouillet et al., 2021, Camos et al., 2011). Articulatory rehearsal is carried out by the articulatory loop without much involvement of attention and executive control, and it becomes an automated maintenance strategy throughout development. In its turn, attentional refreshing consists in the rapid switching of attention between processing and storage that can take place depending on the cognitive load. Contrary to articulatory rehearsal, it is heavily dependent on executive control. However, the authors do not attribute the control of this switching mechanism to a central executive component in the model. They uphold that executive control is an emergent property of goal-directed behaviour instead of an operation of a central executive (Barrouillet & Camos, 2021).

By positing two independent maintenance mechanisms in working memory, the TBRS model offers adequate theoretical accommodation to common phenomena in the verbal domain (e.g., the word length effect, the phonological similarity effect) and to the observation that switching between cognitive tasks incurs in costs to working memory performance (Liefooghe et al., 2008) without assuming the existence of a homunculus (Lemaire et al., 2018). As we previously mentioned, in their last conceptualization of the multicomponent model, Baddeley and al. (2021) acknowledged attentional refreshing as a process acting upon the contents of the visuospatial sketchpad and the episodic buffer.

Similarly, other authors who have proposed their own theoretical models endorse the view that attentional grabbing activities and the rapid switching of attention between stimuli and/or memory representations are core variables for maintenance in working memory (Cowan, 2021; Mashburn et al., 2021; Rhodes & Cowan, 2018; Unsworth & Engle, 2007; Vandierendonck, 2014, 2021).

1.1.3. The embedded-processes model of working memory

The embedded-processes model is a non-modular model of human information processing (Cowan, 1988; 1999), in which working memory is conceptualized as "the ensemble of components of the mind that hold a limited amount of information temporarily in a heightened state of availability for use in ongoing information processing" (Cowan et al., 2021, p.45). This definition sets a marked theoretical difference in comparison to the multicomponent and TBRS models. In the embedded-processes model, working memory is regarded as a set of coordinated mechanisms ("the ensemble of components of the mind") acting to prioritize mental representations in one's consciousness during a task, rather than an architectural feature of human cognition, such as a store and/or separate memory system. Memory is a unitary construct with no modular stores based on the durability of memory traces, but rather a system where information can assume different levels of accessibility. The content of working memory is the subset of semantically interpreted information from long-term memory that is currently activated under the focus of attention and available to conscious manipulation.

The embedded-processes model was proposed by Cowan (1988) as an alternative view to multistore models of human processing based on Broadbent's pipeline account (Broadbent, 1958). A short digression is needed here. Broadbent's information processing model influenced theories of both attention and memory by setting forth two important notions. The first is that attention acts as a selecting mechanism of perceptual information accessing conscious awareness and higher order memory stores. The nature of this selecting mechanism was greatly discussed – a filter of sensory input, an attenuator of sensory representations, a spotlight or zoom lens in the visual field (Deutsch & Deutsch, 1963; Eriksen & St. James, 1986; Fernandez-Duque & Johnson, 1999; Moray, 1959; Norman, 1969; Posner & Petersen, 1990; Posner et al., 1980; Treisman, 1969). The second is that information is sequentially transferred from lower to higher level memory stores (beginning at the sensory register upwards to long-term memory), and that a series of

controlled processes take place to regulate the permanence and exchange of information between the stores (e.g., encoding, retrieval, recoding, rehearsal). Although Broadbent's filter theory was later dismissed by evidence and replaced by other models of human processing, its gist was absorbed by theories and remains influent in contemporary cognitive psychology.

The main critique made by Cowan (1988) to Broadbent's multistore model regards the ordering of the information flow through the memory stores, after passing through the attentional filter. For instance, he argues that pattern recognition and object coding must occur before information enters the short-term store. Both processes rely on long-term knowledge, but long-term memory is reached only later in the multistore model. Possible exceptions for the need of long-term knowledge in encoding information to a short-term store are innate feature detectors, but they are tuned by experience in early childhood. Infants quickly learn phonetic categories from their speech environment (Werker & Tees, 1984) and visual perception develops from partial to whole representations of objects from 3 weeks to 4 months of life (Aslin & Smith, 1988). Another example regards priming effects. Classic studies show that lexical decisions (therefore based on long-term knowledge) are influenced by semantic content presented just below the critical threshold for awareness (i.e., the stimuli affect memory performance even if participants are not aware of them) (Balota, 1983; Jacoby & Whitehouse, 1989). Because the multistore model assumes that awareness is on the contents of the short-term store, priming effects cannot be explained by a model assuming that information enters long-term memory *after* passing short-term store, where lies awareness.

Cowan et al. (2021) summarize the problem as follows: 1) if the content of working memory includes not only newly-encoded sensory information, but also prior knowledge allowing one to identify/interpret information (e.g., object identification, speech), then it is reasonable to assume that encoding in working memory *depends* on contact with long-term memory; 2) yet, new information often must be held in working memory *before* it can form new long-term memory traces (my italics). To account for this "apparent paradox" (Cowan et al., 2021; pp. 48), the approach of multistore models is to place bidirectional arrows representing the transit of information between short-term and long-term stores (i.e., modules within the system). Instead, Cowan proposes a graphic representation (Figure 3) where information in memory forms subsets of elements based on their level of activation. The largest subset is long-term memory, whose most of the content is at a low

state of activation during a given task. A second subset is formed by elements of long-term memory that are activated during the task; this consists in short-term memory as referred by modular models. Finally, a narrower subset is formed by the elements in the focus of attention, where conscious awareness takes place. The focus of attention deals only with a limited number of memory representations at any given time (Cowan, 2001; Cowan et al., 2005). These representations are the items about which a person is currently "thinking". Therefore, they correspond to the content being processed by working memory if we use the modular nomenclature.

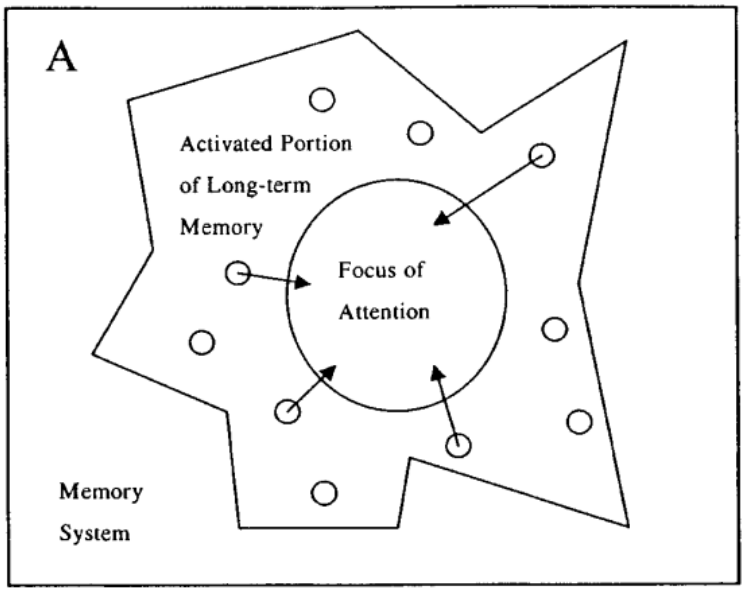

Figure 3. The embedded-processes model of working memory (reproduction from Cowan, 2001). The arrows represent activated information entering the focus of attention.

Conceptually, working memory is the ensemble of mechanisms acting to keep information in a heightened activation state. The maintenance of information in this activated state is made by rehearsal and attentional refreshing. This implies that, in the embedded-processes model, temporal decay in working memory is a function of the level of activation of long-term memory. Also, because all information is assumed to be a subset of long-term memory, the transient state of high activation in the focus of attention is a function of memory consolidation. Poorly consolidated traces decay more quickly, whereas well consolidated traces last longer in the activated state (e.g., a novel, easy to forget, sequence of digits versus a familiar telephone number).

Albeit acknowledging the different codes (spatial, verbal, etc.) and sensory modalities (touch, visual, auditory, etc.) reaching memory, the model focuses on the similarities on how different types of information are processed and maintained on the system. This is the reason why the graphic representation of the model does not include modality-specific sensorial buffers nor an episodic buffer. However, the model includes two modules flanking long-term memory: a brief sensory store and a central executive. The first registers sensorial input in the brain and causes a fleeting sensation for few hundred milliseconds. This sensory store is analogous to iconic (Sperling, 1960) and echoic memory (Neisser, 1967, as cited by Norman, 1968). Stimulus input coming from the sensory store activates the elements of long-term memory based on their perceptual features (e.g., intensity, salience, constancy). An attentional filter is not required in the model because unchanged perceptual stimuli do not recruit attention by effects of habituation. However, if a perceptual change happens, the habituated stimulus will recruit attention and orientation, thus accessing the focus of attention. The absence of a filtering mechanism between the sensory store and long-term memory accounts for the cocktail party effect (Cherry, 1953)[1], another paradox in Broadbent's model. The central executive, in its turn, deliberately controls attention and orients its focus to achieve goals. The controlled processes carried out by the central executive include guided retrieval from long-term memory (i.e., the effortful use of introspective attention to recall information) and the voluntary orienting of attention to sensorial input. In neural representational terms, the central executive assembles the activity of the frontal lobe and areas in the parietal lobe.

Besides the absence of specific storage modules, we highlight a second major difference of the embedded-processes model compared to modular memory models. Attention is not seen as a filter or gateway to consciousness and the successive encoding of memory traces (Broadbent), nor as an energetic resource for controlled processes (multicomponent and TBRS models). Continuing with the metaphors, it is rather seen as *stage* where memory representations are attended and displayed to conscious awareness.

[1] The cocktail party effect is a selective hearing phenomenon in which one focuses attention and isolates a single auditory message while filtering the background noise. As in a noisy party, people can focus on one conversation despite the multitude of acoustic stimulation in the room, yet certain background stimuli can break the attentional filter and are consciously perceived by the individual (e.g., hearing your own name being mentioned in a conversation nearby, a sudden change in the musical playlist, a glass breaking accidentally, etc.). In his experiments, Cherry (1953) employed a dichotic listening paradigm in which participants are required to listen to two different messages in each ear via headphones and to isolate one of them, by repeating it aloud.

Once outside the focus of attention, short-term information goes to the backstage of the system (long-term memory) until it is activated again.

To conclude this section of the chapter, we draw some general comparisons between the models presented herein. Both modular and non-modular models suggest that maintenance in working memory relies on attentional processes, but each model proposes its specific view on how attention works to maintain information and on the type of material it can be employed. For the embedded-processes (Cowan, 1988, 1999) and the TBRS model (Barrouillet et al., 2004; Barrouillet & Camos, 2015), attention-based maintenance can be applied to any type of information, disregarding sensorial modalities and representational codes. For the multicomponent model (Baddeley & Hitch, 1974; Baddeley & Logie, 1999; Baddeley et al., 2011), it is mainly applied to multimodal information stored in the episodic buffer. However, both the TBRS and the multicomponent models suggest that subvocal articulatory rehearsal is the specific maintenance mechanism for verbal information. The embedded-processes model acknowledges also the existence of subvocal rehearsal as a maintenance mechanism.

Despite the particularities mentioned above, the models agree on the fact that memory for visuospatial material is relying on attention. Hence, considering the current state of the theoretical debate, the best experimental strategy to investigate the role of attention in working memory is by using visuospatial material. Nevertheless, this thesis will also test the role of attention on verbal material as the TBRS predicts that it should also depend on attentional refreshing. Next, we will present data on the development of working memory, its capacity limits, and its functioning mechanisms during life, noticeably the acquisition of attentional control during childhood.

1.2. The development of working memory in typically developing individuals

Working memory is a capacity limited system, meaning that it cannot hold and manipulate an infinite amount of information at the same time. It can hold information only for a finite period, after which memory traces decay, and can disappear. Likewise, attention can only be directed to a limited number of items and/or mental operations.

Other limitations in working memory regard encoding[2], processing speed[3], retrieval[4], and response selection[5]. These capacities and constraints are not the same across all ages. In this section, we will address how they change throughout the lifespan, focusing on the optimization of the mechanisms that support the storage of information during childhood.

1.2.1. The age-related increase in working memory capacity

From infancy to adulthood, working memory undergoes quantitative and qualitative changes. The quantitative changes in working memory regard the increase in the amount of information therein (i.e., the number of items, chunks, or slots of complex visual objects) and the increase in the duration of memory traces. In their turn, the qualitative changes regard the maturation of its operating mechanisms. Here we will focus on the increase in capacity, the optimisation of attentional control, and the acquisition and development of maintenance strategies that counteract temporal decay, namely phonological rehearsal and attentional refreshing. Because the term "capacity" is ambiguously used to refer to both time and storage constraints in working memory, here we will use the expressions "time limits" to refer to the former and "storage capacity" to the latter.

The capacity of working memory increases from infancy to adolescence, peaks around age 20, and then progressively declines until old age (Bopp & Verhaegen, 2005; Brockmole & Logie, 2013; Dempster, 1981; Hester et al., 2004; Simmering, 2012; Wingfield et al., 1988). The first marked period of increase happens during the first year of life. Most research on this period use visual material and measure ocular fixations as behavioural correlates of memory functioning[6]. There is evidence that infants can hold one isolated visuospatial information (e.g., a face, a colour, a spatial location) from the age of 5 to 6 months (Fagan III, 1973; Oakes et al., 2011; Pelphrey et al., 2004; Ross-Sheehy et

[2] For instance: stimulation above a certain input rate is not encoded to working memory, competing stimulation is disruptive to performance, and attentional limitations can yield errors in encoding (Treisman & Schmidt, 1982; De Fockert et al., 2001; Kelley & Lavie, 2011).

[3] It means that one can only perform a given mental operation if proper time is warranted (Case et al., 1982; Chi & Gallagher, 1982).

[4] Retrieval is a time-consuming process involving a controlled search and selection of items in the memoranda (Rosen & Engle, 1997; Townsend & Fifić, 2004; Unsworth, 2008, 2009).

[5] The type of response required by a memory task can be a bottleneck to performance (Pashler, 1991; Towse, 1998).

[6] The rationale is based on the evolutionary preference of human infants for novelty: babies tend to spend more time looking to novel visual stimuli, which implies habituation and rudiments of memory representations (Fantz, 1964; Friedman, 1972; Slater et al., 1982, 1984).

al., 2003). This capacity rapidly increases within months. For instance, Oakes et al. (2006) showed that 6.5-month-old infants can represent both colour and location separately, one feature at a time, in visuospatial short-term memory[7]. By the age of 7.5 months, they could make colour-location bindings as effectively as their 12.5-month-old peers.

Memory for complex physical objects (e.g., a toy) also increases during the first year of life. Diamond (1985) showed that by the age of 9 months, infants can recall two complex objects, and Kibbe and Leslie (2011) showed that by the age of 6 months they can notice the absence of an object hidden by a display. Before that, babies stare randomly after the "disappearing" of a hidden object, which suggest that they do not store memory traces about the object's location (Reznick et al., 2004). Regarding the time limit of memory traces in working memory between 6 and 12 months, studies suggest an increment of 2 seconds per month (Schwartz & Rerznick, 1999).

A second period of increase in working memory capacities have been evidenced during childhood, when investigation on the phonological domain becomes possible. In a literature review, Dempster and colleagues analysed the span measures for digits, letters, and words in children with different ages. They showed a constant increase in from the age of 2 to 12 years old. For instance, five-year-old children have a mean span of nearly 2.8 one-syllable words, whereas seven-year-olds have a mean span of 3.75, and eleven-year-olds have a mean span of 4.8 (Hitch et al., 1989, Experiment 2).

Gathercole and colleagues (2004) found a similar pattern using neuropsychological assessment tools in a sample of children from 4 to 15 years of age. They assessed each component of Baddeley and Hitch's (1974) working memory model (i.e., the phonological loop, the visuospatial sketchpad, and the central executive) and found a similar linear increase of performance on all measures as a function of age. Their data also suggest that the tripartite structure of working memory (i.e., phonological loop, visuospatial sketchpad, and central executive, as proposed by the multicomponent model) is already present at age 6. Accordingly, measures of verbal and visuospatial working memory do not correlate at the age of 5 years (Michas & Henry 1994), and 8 years (Liberman et al., 1982; Pickering et

[7] We reproduce the term used by the authors, "visuospatial short-term memory" instead of "working memory", because the processing component of working memory is not directly tapped by the visual change paradigms used with infants. Although the terms "working memory" and "short-term memory" are sometimes used interchangeably in the field, for conceptual uniformity in the thesis, here we use the expression short-term memory thoroughly when talking about paradigms that tap only the storage component of working memory.

al., 1998): this provides another evidence for the dissociation of verbal and visuospatial working memory from early age.

Specifically, in the visuospatial domain, the storage capacity for visual information increases more rapidly than the storage capacity for spatial sequences during childhood (Logie & Pearson, 1997). In their study, Logie and Pearson (1997) assessed memory for visual patterns (two dimensional arrays with certain cases coloured in black) and sequences of movements (the Corsi blocks task) in groups of children of different ages (5-6, 8-9, and 11-12). Memory for visual patterns was better than movement sequences in all age groups and the span measures of both tasks increased linearly from age 5 to 12. However, the rate of increase in the visual task was higher than in the spatial task. These different rates are taken by Logie and Pearson (1997) as further evidence for the separation of the visuospatial sketchpad into an inner scribe and a visual cache in the multicomponent model of working memory (see section 1.1.).

Around age 12, working memory storage capacities are almost fully developed (Luciana & Nelson, 2002), but the efficacy of its functioning will be optimized until the end of the adolescence. The maturation of the frontal lobes – that develop until age 30 (Huttenlocher, 1979) – plays an important role in this optimization. Performance in tasks that are more strongly reliant on the prefrontal cortex keep progressing after the age of 12, opposed to those supported by more posterior neural substrates (Conklin et al., 2007). For example, recognition memory for verbal material and faces reaches optimal performance around age 9 (Conklin et al., 2007), and performance for the recall of isolated spatial locations develops until age 11-12 (Luciana et al., 2005). In their turn, the ability to concurrently maintain and manipulate multiple spatial locations (e.g., a backward spatial span task, such as the reverse Corsi blocks test) progress until age 13-15, and performance on tasks demanding the strategic organization of the memorized content keeps progressing until age 16-17. Hence, adolescence is a period of optimization of the executive control of working memory operations.

All the changes abovementioned cumulate in an adult storage capacity of around 5 verbal items that can be improved to up to 8 items depending on the type of strategy employed (Barrouillet et al., 2021). In the visuospatial domain, the storage capacity of an adult is limited to around four individual features (e.g., color, spatial orientation, shaded textures) and four to five integrated objects (Alvarez & Cavanagh, 2004; Awh et al., 2007;

Cowan, 2001; Luck & Vogel, 1997; Todd & Marois, 2004), with some disagreement in the field on whether there is a fixed number of slots to hold unitary representations or a flexible limit depending on items' visual complexity, salience, the task's goals, and attentional allocation (Bays & Husain, 2008; Bays et al., 2011; Hardman & Cowan, 2015; Lilburn et al., 2019; Ma et al., 2014; Schneegans & Bays, 2016). Some recent discussions also include the precision of representations in visuospatial working memory as a constraining factor of its storage capacities (Lilburn et al., 2019; Roggeman et al., 2014; Xie & Zhang, 2017).

The transition from visual to verbal codes is another instance of the debate on the nature of memory representations and their impact on capacity limits. Many authors have suggested that children prioritize verbal codes over visual codes as they grow older, this change happening around age 7 and becoming predominant until age 10 (Al-Namlah et al, 2006; Hitch et al., 1988; Hitch et al., 1991; Logie et al., 2000; Palmer, 2000; Tam et al., 2010). Younger children, on the other hand, are more reliant on visual codes (Brown 1977; Hayes & Schulze, 1977). The preference for verbal codes by the end of childhood would dramatically increase the storage capacity of working memory.

In conclusion, working memory develops from infancy to adulthood, first increasing its capacity limits and lately optimizing its operating mechanisms. Infancy and childhood are periods when the storage and time limits of working memory expand dramatically: a developing child can hold more information in mind and for longer periods with age. Around age 12, a child achieves adult levels of storage capacity, but adult levels of functioning are only reached during the following years. Then, adolescence is the period when individuals become more proficient in maintaining, processing, and strategically manipulating information held in working memory. Of important notice is the fact that the architecture of working memory, as described in the multicomponent model (i.e., phonological loop, visuospatial sketchpad, and central executive) exists from an early age, before the age of 5 (Gathercole et al., 2004), and remains stable during human development. It is the maturation of the brain and the improvement of its processes and interacting subsystems during the lifespan that drive the development of this prior structure. The following section is dedicated to the sources of working memory development during the lifespan.

1.2.2. The sources of working memory development

Many accounts have been invoked to explain the development of working memory. They include the speed of degradation of memory traces, the speed and efficiency of information processing, the increase of cognitive resources, and the acquisition and use of maintenance strategies. As these accounts have been extensively reviewed elsewhere (see Barrouillet & Camos, 2022 and Camos & Barrouillet, 2018), we will present a selection of what is important to our research topic. Our selection emphasizes the growth in attentional resources and in the control over mental operations because the TBRS model that serves as theoretical background to our work has precise predictions with this regard which guide our experimental reasoning throughout this thesis.

1.2.2.1. The increase of cognitive resources. This putative source of working memory development has been proposed by some neo-Piagetan authors (Halford 1993; Pascual-Leone, 1970) and later adopted by contemporary authors (Barrouillet et al., 2004; Bayliss et al., 2005). According to Pascual-Leone (1970), cognitive development is driven by an increase in the number of schemes that can be simultaneously maintained and coordinated in working memory. Schemes are conceptualized as sets of basic knowledge about entities or situations, including their features, parts, properties, and their relations. They allow the interpretation of the sensorial world, the organization of abstract knowledge, and the manipulation of mental representations during mental operations; therefore, they are essential for the maintenance and processing of information in working memory. In Pascual-Leone's theory (1970), it is the growth of the so-called "space M" during childhood that allow children to perform more complex tasks requiring the coordination of an increasing number of schemes, and to store and recall more memory representations.

More recently, other authors evoked the growth in cognitive resource as an explanation for age differences in working memory spans (Barrouillet et al., 2009; Gaillard et al., 2011; Gavens & Barrouillet, 2004). In their experiments, they carefully controlled the time available for processing concurrent information during complex span tasks. In these tasks, children were required to memorize series of letters and to perform concurrent operations in-between each letter. These operations varied from simply reading aloud a sequence of digits to performing arithmetical calculations during variable delays. The authors predicted that if processing speed increases with age and boosts working memory

performance, by weighting the time devoted to processing across the age groups according to their needs (thus equalizing the cognitive load of the task, as defined by the TBRS model), then the group differences in the span measures should disappear. Although this manipulation drastically reduced the age differences in performance, a residual effect of age remained, with older children achieving higher memory spans. The authors propose that this residual age effect on recall performance reflects the growth of cognitive resource.

Results from a study by Cowan et al. (1999) also support the idea of an increase in cognitive resource underlying the development of working memory. The study required adults, eleven-year-olds, and seven-year-olds to memorize sequences of digits while playing a video game. The number of digits correctly recalled augmented with age despite the length of the sequences. According to the authors, this is due to an increase in the number of chunks that can be simultaneously maintained in working memory under the focus of attention. More recently, Gilchrist et al. (2009) tested the alternative hypotheses that an increase in the size of the chunks (i.e., the amount of information in a single chunks) drives working memory development. They presented different types of lists of words (short sentences, long sentences, and non-related words) to adults, seven and twelve-year olds. The younger group recalled less sentences than older children and adults, but there were no age-related differences in the number of words correctly recalled from the sentences. The authors concluded that there is no expansion in the size of the chunks stored in working memory, but rather an increase in the number of chunks that can be stored (i.e., the number of words forming a meaningful unity that can be recalled did not change with age). This expansion in working memory storage capacity in the form of chunks could be related to a general growth in cognitive resources.

Even though a developmental growth in cognitive resources might be plausible, this presumptive source of working memory development is not supported by direct evidence. This is because the concept of cognitive resource is somehow vague and difficult to be translated to experimental settings. For example, measures of working memory capacity can be taken as a measure of the amount of the "cognitive resource" available in the system (the causal variable under investigation), leading to circular reasoning. As exposed above, studies suggesting the developmental increase of cognitive resource often rely in effects that are not directly tapped by their experimental design. Next, we will explore another potential source for the development of working memory whose investigation is based in experimental tasks other than the complex span paradigm.

1.2.2.2. The growth of the focus of attention. The embedded-processes model (Cowan, 1998, 1999) proposes that the content of working memory consists in activated long-term representations on the focus of attention. A limited attentional focus, therefore, is a constraining factor of working memory capacities (Cowan, 2001). As a logical consequence, an age-related growth of the focus of attention is a potential drive for the development of working memory.

To test this hypothesis, Cowan and collaborators have estimated the size of the attentional focus and its progress throughout childhood by using different paradigms. The first is a change detection paradigm. In this type of task, a set of visual stimuli is briefly presented and then followed by a second set; the participants must compare the two sets and answer whether there was a change between the stimuli in the first and in the second set. Because the presentation delay of the first set is very short, verbal encoding or chunking strategies are prevented and performance is thought to reflect a pure measure of working memory storage capacity. It is then possible to estimate the size of the focus of attention by manipulating the size of the set of stimuli: once performance drops contingently to an increase above *n* elements in the set, the maximum number of elements (*n*) in the focus of attention is considered to have been reached. The second paradigm is the running span, in which participants are exposed to a rapid aural presentation of lists of numbers of different lengths. The lists are interrupted unpredictably, and participants are required to recall many items as they can from the end of the list. Again, the rapid presentation and the unpredictability of the task renders memory rehearsal difficult, and recall performance is considered to reflect the content of the attentional focus (Bunting et al., 2006). Through these tasks, Cowan et al. (1999) proposed that the size of the focus of attention increases from 2 items around the age of 6-7 years to 4 items in adulthood, four being the maximum number of chunks in the attentional focus of a typically developing adult.

The number four had already been proposed by Halford et al. (2005) as a limiting factor of cognitive development. In a series of reasoning tasks, they showed that four is the maximum number of variables that adults can simultaneously process while solving a problem. Above five variables in a problem, their performance dropped to chance level. In his theory (Halford, 1993), human reasoning is based on relational mental models that are acquired via experience. These mental models serve as structures to store and manipulate information, for instance, representing hierarchical inclusion or exclusion, categorical knowledge, or understanding of quantity. From childhood to adulthood, individuals

acquire more complex relational structures by integrating dimensions and/or elements to a mental model. One's ability to reason depends on the mental models available, and the complexity of a mental model is a function of the number of elements in the model. Interestingly, the maximum number of variables in a mental model corresponds to the estimated size of the attentional focus in working memory, that is, four chunks (Cowan, 2001; Halford et al., 2005). Halford et al. (2007) argument that these similar limitations in working memory and reasoning reflect a central capacity limit in human cognition and probably a shared demand on attention.

Besides the number of elements in the focus of attention, the increasing control over this focus also plays a role in the development of working memory. Cowan et al. (2006a) orthogonally manipulated the size of the focus of attention and the control of attention in a series of serial recall and change detection tasks. Their results showed that these measures are highly correlated among adult participants, but not in children (eleven-year-olds), which indicates a possible independence between mechanisms.

The ability to use attention to inhibit task-irrelevant stimuli is also an aspect of attentional control that develops with age (Bjorklund & Harnishfeger, 1990). Maccoby and Hagen (1965) developed a task to test the effect of distraction upon memory for task-relevant or irrelevant material. Children from first, third, fifth and seventh grades were instructed to memorized the background colour of cards depicting objects and animals for later recall. At the end of a trial sequence, children were shown a set of cards all in the same background colour, and asked to answer which card had the correct match of picture and background colour seen on previous trials. A group of children performed the task without distraction while another had to simultaneously judge the pitch of musical notes. Recall for task-relevant material (the background colours) increased regularly with age whereas recall for task-irrelevant material (the match background-pictures) remained stable from first to fifth grades, then declined from fifth to seventh grades. The distraction task lowered performance for task-relevant material but it had no impact on memory for task-irrelevant material in all age groups. These results suggest that the ability to inhibit task-irrelevant material and focus attention on relevant stimuli grows with age and is damaged by distraction.

To summarize, both the size of the focus of attention and attentional control increase with age during childhood. From starting elementary school, children will see the

number of chunks they can attend and the number of variables they can simultaneously consider during reasoning to augment. Both will reach a plateau of four chunks and variables in adulthood. As for the attentional control, children become increasingly capable of supressing distractors and irrelevant information in working memory, yet this ability remains prone to disruption by other attentional-demanding tasks, even in adults.

1.2.2.3. The emergence of new strategies and their efficiency. Another source of working memory development is the improvement in the ability to voluntarily use and coordinate maintenance strategies to counteract memory decay. Following the gradual increase of the storage capacity during early childhood, presented in section 2.1., qualitative changes in working memory happen near the transition between preschool and school-age. Around age 7, children will first use verbal repetition as a maintenance strategy and then will gradually employ attentional refreshing, a strategy that will mature until adolescence (Allik & Siegel, 1976; Barrouillet et al., 2009; Camos & Barrouillet, 2011; Flavell et al., 1966; Halliday et al., 1990). These two maintenance strategies will develop independently throughout the life span (Camos et al., 2009; Mora & Camos, 2013, 2015; Oftinger & Camos, 2016, 2018), with phonological rehearsal having a constant capacity during the life course (Hitch et al., 1989) and attentional refreshing maturing until adolescence (Barrouillet et al., 2009). We first explore the emergence of verbal repetition, and we will later address the use of attentional refreshing in a separate section.

The use of phonological rehearsal. In one of the first studies on children's mnemonic strategies, Flavell et al. (1966) showed that kindergarteners do not engage in overt verbal rehearsal[8] during a recall task. Older children (second graders, aged 7-8 years, and fifth graders, aged 10-11 years), in their turn, uttered the name of stimuli more often and had higher memory performance. This use of verbal repetition depends on the acquisition of verbal codes and the progressive internalization of speech during childhood. Flavell et al. (1966) commented with surprise that even second and fifth graders engaged in overt verbal rehearsal during the task, contrarily to Vygotsky's (1966, as cited by Flavell et al., 1966) hypothesis of the complete internalization of non-social speech during early childhood. The authors defend that a better conceptualization of "inner speech" is needed.

[8] Overt verbal rehearsal is the action of repeating a word aloud and/or uttering the word silently only with lip movements. It refers to the observable motor behaviour of repeating a word, as opposed to covert verbal rehearsal. In the latter, the individual engages in subvocal repetition by recruiting circuits perceptual and motor brain circuits involved with speech comprehension and production, however without producing a motor response (observable overt behaviour).

In any way, their results showed that children progressively become more reliant in verbal rehearsal as their memory spans increase. Later, other studies later showed that the use of articulatory rehearsal becomes more frequent during childhood, optimizing verbal working memory (McGilly & Siegler, 1989; Ornstein & Liberty, 1975).

The absence of verbal rehearsal in younger children raises the question on whether they are capable of doing it or whether they simply do not use this strategy by lack of metacognitive skills. It could be the case that, although capable of performing this strategy, younger children do not realize how efficient verbal rehearsal is. Hagen et al (1973) tested this hypothesis by explicitly instructing five-year-olds to use verbal repetition during recall tasks. Not only receiving the instruction had no impact on performance, but also children would stop using verbal rehearsal unless prompted by the experimenter. Also, there no improvement resulting from the rehearsal instructions was observed in a one-week follow up, which demonstrate that participants did not learn rehearsal strategy.

Verbal rehearsal is a very efficient strategy to maintain the order of elements in a list. In a sample of eight- to ten-year-olds, Lehmann and Hasselhorn (2007) showed that older children tend to use rehearsal in a cumulative fashion (i.e., by repeating the sequence of words from the beginning) more often than their younger peers. In their study, they distinguished three different maintenance strategies used in a verbal recall task. The first strategy was labeling, when children spoke the name of an item only once during the interstimulus interval (e.g., "cat"). The second was single-word rehearsal, that is, when children repeated several times the name of one item (e.g., "cat, cat, cat"). Finally, cumulative rehearsal corresponded to the repetition of at least two items (e.g., "cat", "fork"). The authors found children's preferred strategy changed according to their age, the length of the lists, and the serial position of the items. Children switched strategy from labelling to cumulative rehearsal as they grow older and as the list length increased. Also, they preferred using cumulative rehearsal to maintain words at early serial positions whereas they used labelling for later serial positions.

The increasing ability to verbally rehearse items during childhood will also positively impact memory for visuospatial material. Cowan et al. (2006b) asked children (9- and 12-year-olds) and adults to perform a binding task where they should memorize sequential associations between names and spatial locations. Participants would see names arrays of three to seven cases on a screen and a name would appear in each case at a time.

They were required to memorize the names and their corresponding places in the arrays. Participants rely in two main strategies to maintain this name-location associations: they can either chunk information or verbally rehearse the names while simultaneously reactivating the positions the array, as if mentally recreating the "path" traveled by the names in the array.

According to the authors, the chunking strategy would reflect an abstract representational code in working memory. The "traveling while repeating" strategy would reflect verbal and spatial codes held separately but used in parallel during rehearsal. To tap the use of these strategies, the authors manipulated the match between names and locations by varying the number of possible associations in a trial. In the 1-to-1 condition, only one name was associated to each location. In the inequal condition, more than one name was presented in the same location. This inequal condition is particularly detrimental to the use of the "travel" strategy. Results revealed a major developmental change of strategy: 9-year-olds had better performance in the inequal condition, 12-year-olds had no difference between conditions, and adults had better performance in the 1-to-1 condition. The authors argument that the emergence of subvocal repetition allows adults to store information in verbal and spatial codes in parallel, thus using the "travel" strategy as they verbally rehearse the names. Contrarily, children who are not yet able to implement verbal rehearsal need to rely in more costly verbal-spatial associations. The fact that the benefit in the 1-to-1 condition was abolished when adults were required to perform articulatory suppression strengthens this interpretation.

As seen above, the acquisition of phonological rehearsal around age 7 contributes to the growth of working memory capacity during childhood. Verbal rehearsal is first implemented overtly, by uttering words aloud, and gradually becomes a silent strategy. There is no evidence that younger children implement verbal rehearsal spontaneously, nor that they benefit from this strategy when instructed to use it. Once the ability to use verbal rehearsal is achieved, children continue to use it until later life, together with another powerful strategy that emerges during childhood.

The use of attentional refreshing. As we presented in section 2.2.2., the increasing focus of attention and the control over it are drives for working memory development during childhood. This enhanced control of attention will allow children to perform attentional refreshing, a strategy highlighted by the TBRS model. Attentional refreshing

consists in the rapid switching of the focus of attention between processing and maintenance activities. During short pauses in processing, one can reallocate attention to the content of working memory and prevent temporal decay by reactivating the memory traces. The main method to study the use of attentional refreshing is through the complex span paradigm. By varying the pace of the secondary task (thus the cognitive load of a task, as defined by the TBRS model), it is possible to constrain the use of attentional refreshing to maintain the memoranda. For instance, in a fast-paced complex span task, the intervals between items are too short for participants to implement attentional refreshing because their attention is constantly captured by the processing of stimuli. The more one relies in attentional refreshing, the more her performance will be damaged by conditions preventing its use. Conversely, the absence of a negative effect in these conditions indicates that participants do not implement attentional refreshing.

Barrouillet et al. (2009) tested this hypothesis by asking 5- and 7-year-olds to memorize sequences of animals and to label the colour of smiley faces in-between each animal in the sequence. The cognitive load of the task varied according to the number of smileys presented during the interval between the animals (0, 2, or 4 smileys). The results showed memory spans of five-year-olds were harmed by the presence of the secondary task, but the number of smileys did not affect performance. Younger children had poorer performance when they needed to judge the colour of the smileys (in comparison to the zero-smiley condition), but no difference between the two and four-smiley conditions was found. On the contrary, the group of 7-year-olds was highly affected by the cognitive load of the secondary task. This indicates that younger children do not implement attentional refreshing or are not yet able to perform it.

Similar results were reported by Camos and Barrouillet (2011). They tested the hypothesis that, if attentional refreshing is not implemented to counteract temporal decay, forgetting will be directly influenced by the duration of the interval between items. They manipulated orthogonally the cognitive load of the secondary task and the duration of the intervals by presenting 1 or 2 smileys during 2 or 4 seconds. The time to implement attentional refreshing is equated in the conditions "1 smiley in 2 seconds" and "2 smileys in 4 seconds" because they have the smiley per second ratio (0.5 smiley/second). If children do not use attentional refreshing, the conditions with four seconds would yield worse performances despite the number of smileys presented. The authors found that the

performance of 7-year-olds depends on the cognitive load of the task, whereas the performance of 5-year-olds depends on the duration of the intervals.

Tam et al. (2010) adopted a similar methodology to test the use of attentional refreshing and verbal rehearsal in 6- and 8-year-olds. They manipulated the nature of the concurrent processing task (verbal or nonverbal), the phonological similarity of the items, and the delay interval between items in a series of Brown-Peterson tasks[9]. Verbal concurrent tasks are deleterious to the use of both strategies, but they have a specific detrimental effect upon verbal rehearsal. Nonverbal processing tasks, in their turn, do not impact verbal rehearsal as much. The authors found that verbal processing was more detrimental to 8- than to 6-year-olds and that nonverbal processing tasks impaired performance in both age groups. This pattern of results allows two major interpretations. First, that the developmental increase in working memory capacity from 6 to 8 years is mainly driven by an enhancement in phonological rehearsal. Second, that nonverbal processing tasks prevents the use of attentional refreshing from age 6.

Another way to investigate the use of attention in working memory is by introducing cues the stages of encoding (pre-cues) and maintenance (retro-cues) of tasks. The latter ones are of particular interest because they retrospectively guide the participant's attention to one item in the memoranda. Retro-cues are, therefore, an experimental way to manipulate attentional orienting to the content of working memory, and any benefit in performance attributed to the presence of retro-cues can be taken as an index of the ability to voluntarily orient attention. A study by Shimi and colleagues (2014) showed that children benefit from retro-cues since age 7 in a color recognition task. Between ages 10-11, the benefit in performance provided by the retro-cues was the same as in adults. This is suggestive that, at least for typically developing individuals, the ability to voluntarily use attention to boost working memory performance is present from age 7 and it is mature by the beginning of adolescence. Moreover, the authors showed in a second experiment that low-validity cues (i.e., cues that indicated the relevant item inconsistently, sometimes indicating the wrong item) did not hinder memory performance for all age groups. Even 7-year-olds were able to ignore deceptive retro-cues during the task. The results strengthen

[9] The Brown-Peterson paradigm consists in presenting a sequence of stimuli for memorisation (originally three-letter sequences called "trigrams"), followed by a concurrent processing task to prevent the usage of mnemonics during the retention interval. Differently than the complex-span paradigm, where participants perform a processing task in-between each item of the sequence, in the Brown-Peterson task, the processing task is presented after the whole sequence of stimuli for memorization.

the evidence in favour of the increasing control of attention and the voluntary nature of attentional orienting in working memory tasks during childhood.

Gaillard et al. (2011) also investigated the use of attentional refreshing from mid-childhood to early adolescence. They asked children of 8 and 11 years to perform a complex span task in which the time available to refresh the items was equated between age-groups and proportional to the processing speed in each group. Because younger children process information more slowly than older children, differences in performance issued by equal time intervals between age groups are prone to reflect group differences in processing speed rather than in the use of refreshing. For this reason, younger children were granted more time to perform attentional refreshing. The additional time yielded an increase in memory performance in 8-year-olds and abolished performance differences in comparison to 11-year-olds. The authors argue that attentional refreshing is performed more slowly by younger children and that a growth in efficacy of this strategy is a potential drive for the age-related working memory development.

All the results presented above suggest that important developmental changes happen around age 7 regarding the emergence of attentional refreshing, as it is for phonological rehearsal. Nevertheless, Bertrand and Camos (2015) noticed that more ecological and appealing experimental settings might promote the use of attentional refreshing by children aged between 4 and 6 years. In a task that emulated a grocery store with plastic fruits, children were asked to memorize a list of items and to "do the shopping like mommy". The concurrent attentional demand was varied by asking children to walk a different path to the grocery shop, with obstacles that also increased the motor complexity of the task. The authors also introduced an unfilled delay between the encoding of the "shopping" list and recall, by telling children to until the opening hour of the grocery shop. They found an overall improvement of memory performance with age; a decrement of performance caused by the delay in all age groups; and a slight increase of performance in the concomitant motor condition ("walk to the shop"). The authors argue that the reduction of performance in the unfilled delayed condition is caused by the children's inability to use attentional refreshing even when they have nothing else to do. They make a caveat, however, that the low-demanding "walk to the shop" condition increased recall. This allows the tentative interpretation that, under certain conditions that favour the maintenance of the task's goal, even 4-year-olds are sensitive to changes of attentional demand and could implement attentional refreshing.

Complementarily to studies with children, research with healthy elderly population has shown that attentional-based strategies remain important during later life, despite the natural decline in working memory capacities. Studies showed that the age-related decline in working memory cannot be exclusively accounted to failures in the voluntary control of attention during memory tasks. Albeit the retro-cue benefit for older adults is not as big as in younger adults, the beneficial effect of retro-cues to boost memory performance is preserved in this age group (Gilchrist et al., 2016; Loaiza & Souza, 2018, 2019; Maniglia & Souza, 2020; Souza, 2016; Strunk et al., 2019, but see also Duarte et al., 2013 and Newsome et al., 2015, for conflicting results). The results demonstrate that healthy elderly population effectively uses the information provided by the retro-cues to enhance their memory performance.

In this section, we presented data on how attentional refreshing is potentially performed by children as young as 4 years, how it emerges around the age of 6-7 years, and how it is optimized until age 10-11. We also explored how this ability is maintained by elderly populations despite the age-related decrease in working memory. Together, this body of knowledge suggests that increasing attentional control in childhood and adolescence is a drive for working memory development and that once the ability to perform attentional refreshing is achieved, the use of this maintenance strategy continues during later life.

Through this chapter, we presented three major theoretical models of working memory and we drew an overview of the development of working memory from infancy to adolescence in typically developing individuals. The emergence of maintenance strategies such as phonological rehearsal and attentional refreshing play a determinant role in the age-related increase of working memory. We stressed data on these two sources of development over other sources described in the literature because they are the two maintenance strategies emphasized by our theoretical framework, the TBRS model. In the next chapter, we will review the literature on children and adolescents with attention deficit/hyperactivity disorder with a focus on their cognitive functioning and specific deficits in working memory.

Chapter 2

The Attention-deficit/Hyperactivity disorder

The attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterized by severe symptoms of inattention, impulsivity and hyperactivity (American Psychiatric Association, 2013). It has 5.29% of prevalence in children around the world (Polanczyk et al. 2007) and estimates ranging from 2.5% to 3.4% amongst the adult population (Fayyad et al., 2007; Simon et al., 2009). Because of its association with low academic achievement, mathematical cognition and reading comprehension (Kuhn et al., 2016; Miller et al., 2013; Zendarski et al., 2017), ADHD concerns not only public health agents but also educators and psychologists. As we will expose further in this chapter, the cognitive profile of children with ADHD includes core deficits in executive functioning and working memory, which underlie the difficulties in school activities that these children face during their education. It is precisely the cognitive profile of children with ADHD what makes the investigation about this disorder a promising avenue to understanding working memory functioning and development. We will start this chapter by presenting the symptomatology of ADHD, its classification according to different diagnostic manuals, its comorbidities, differential diagnosis, and course during the lifespan. Data about the cognitive profile of children affected by ADHD will then be presented. We will conclude by outlining the main theoretical models of ADHD functioning from the scientific literature, upon which the rationale of this thesis was built.

2.1. Symptomatology

ADHD is a clinical condition composing the group of neurodevelopmental disorders listed in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5, American Psychological Association, 2013). In the International Statistical Classification of Diseases and Related Health Problems (ICD-10, World Health Organization, 2015), it is named hyperkinetic disorder (disease code F90.-) and it is categorized amongst the "behavioural and emotional disorders with onset usually occurring in childhood and adolescence". For the remaining of this thesis, we will adopt the nomenclature from DSM-

5 and mention the term "hyperkinetic disorder" only when discussing specific propositions of the ICD-10[10].

The core feature of ADHD is a persistent pattern of inattention and/or hyperactivity-impulsivity that hinders the patient's normal functioning and development. The behavioural display of inattention can be exemplified by a lack of persistence during tasks, some difficulties in sustaining focus, disorganization not attributable to a lack of understanding or defiant behaviour, and straying from an activity or conversation goal/subject. Inattention symptoms are usually noticeable during activities implying cognitive involvement or effort, with children typically tending to switch from one activity to another and leaving either without completion. The hyperactivity symptoms refer to an exaggerated motor activity or talkativeness in inappropriate contexts (e.g. a child fidgeting, tapping, running or excessively chatting during a class). The impulsivity refers to one's inability to refrain actions based on forethought, which may lead to harmful conducts (e.g. abruptly crossing the street, getting into physical conflict); it may also manifest as social intrusiveness (e.g. interrupting other when in a conversation) and incautious decision-making (e.g. not reflecting about long-term consequences of a certain action). The hyperactive-impulsive feature of ADHD usually manifests as a poorly modulated and disorganized behaviour, resulting in children displaying a certain "clumsiness". These behaviours are more noticeable in structured situations that demand high levels of self-control, such as attending to a class or a meeting.

The pattern of inattention and hyperactivity/impulsivity is pervasive over different situations and persistent over time, meaning that they usually linger throughout school years and sometimes adulthood and old age (Biederman et al., 2010; Semeijn et al, 2016). Depending on the definitions adopted for remission and persistence, longitudinal studies have shown that ADHD symptoms persist in approximately 65% of the diagnosed cases until age 25 (Barkley et al. 2002; Faraone et al., 2006), albeit their behavioural

[10] The World Health Organization approved the 11th revision of the ICD (ICD-11) in 2019 and put it into effect from January 2022. Because a transition between the use of the 10th and the 11th revision of the ICD is currently underway and the medical literature so far reflects the classifications in the ICD-10, we will not discuss the propositions of the ICD-11 in this chapter. Just to mention, after decades of a nomenclature gap, the ICD-11 adopted the term "attention deficit hyperactivity disorder" as already advocated by the American Psychiatric Association (APA) since the 1980's in older versions of the DSM. As an effect of a convergence between the two manuals regarding the neurodevelopmental disorders, a growing uniformity among the medical community regarding ADHD is therefore expected for the next years.

manifestation can change during the development, with hyperactive and impulsive symptoms declining with age at a higher rate than inattentive symptoms (Biederman et al., 2000; Larsson et al., 2006). For example, hyperactivity is less visible during adolescence and impulsivity can be manifested through rage outbursts and low resistance to frustration (Kieling & Rhode, 2010). In adults, the excess of motor activity is commonly replaced by covert behaviours such as feelings of impatience and restlessness, or can be exhibited by a person seeking busy, agitated workspaces, or even by misconduct in traffic (Kieling & Rhode, 2010; Weiss & Weiss, 2004). Despite their temporal persistence, many individuals learn how to cope with ADHD symptoms and show a gradual improvement on attentional control and a reduction of overactivity over time, either because they learn coping strategies, have the support of family, school teams or partners, or because of the normal prognosis of the disorder. However, authors are careful in asserting that the remission of symptoms is a natural part of ADHD's course, since different rates of reported remissions may instead reflect the variability of definitions adopted in the literature (Biederman et al., 2000; Faraone et al., 2006).

For diagnostic purposes, the first criterion (Criterion A) of DSM-5 is the presence of at least six symptoms from a list of inattention symptoms and/or at least six symptoms from a list of hyperactive-impulsive symptoms, for a minimum 6 months. For teenagers and adults, only five symptoms are required. The frequency and intensity of the manifestation of the symptoms must be inconsistent with the child's developmental level and must cause significant impairment in her social and/or academic activities. Also, they cannot be accounted to oppositional behaviour, defiance and hostility, nor a failure in understanding tasks' requirements and instructions. The lists of inattentive and hyperactive-impulsivity symptoms are presented in Table 1, in *ipsis litteris* citation from the DSM-5:

Table 1.

Inattentive and hyperactive-impulsive symptoms of ADHD according to the DSM-5

Inattentive symptoms (Criterion A1)	Hyperactive-impulsive symptoms (Criterion A2)
a. Often fails to give close attention to details or makes careless mistakes in schoolwork, at work, or during other activities (e.g., overlooks or misses details, work is inaccurate). b. Often has difficulty sustaining attention in tasks or play activities (e.g., has difficulty remaining focused during lectures, conversations, or lengthy reading). c. Often does not seem to listen when spoken to directly (e.g., mind seems elsewhere, even in the absence of any obvious distraction). d. Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (e.g., starts tasks but quickly loses focus and is easily sidetracked). e. Often has difficulty organizing tasks and activities (e.g., difficulty managing sequential tasks; difficulty keeping materials and belongings in order; messy, disorganized work; has poor time management; fails to meet deadlines). f. Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (e.g., schoolwork or homework; for older adolescents and adults, preparing reports, completing forms, reviewing lengthy papers). g. Often loses things necessary for tasks or activities (e.g., school materials, pencils, books, tools, wallets, keys, paperwork, eyeglasses, mobile telephones). h. Is often easily distracted by extraneous stimuli (for older adolescents and adults, may include unrelated thoughts). i. Is often forgetful in daily activities (e.g., doing chores, running errands; for older adolescents and adults, returning calls, paying bills, keeping appointments). (American Psychological Association, 2013, p. 59)	a. Often fidgets with or taps hands or feet or squirms in seat. b. Often leaves seat in situations when remaining seated is expected (e.g., leaves his or her place in the classroom, in the office or other workplace, or in other situations that require remaining in place). c. Often runs about or climbs in situations where it is inappropriate. (Note: In adolescents or adults, may be limited to feeling restless.) d. Often unable to play or engage in leisure activities quietly. e. Is often "on the go," acting as if "driven by a motor" (e.g., is unable to be or uncomfortable being still for extended time, as in restaurants, meetings; may be experienced by others as being restless or difficult to keep up with). f. Often talks excessively. g. Often blurts out an answer before a question has been completed (e.g., completes people's sentences; cannot wait for turn in conversation). h. Often has difficulty waiting his or her turn (e.g., while waiting in line). i. Often interrupts or intrudes on others (e.g., butts into conversations, games, or activities; may start using other people's things without asking or receiving permission; for adolescents and adults, may intrude into or take over what others are doing). (American Psychological Association, 2013, p. 60)

As all neurodevelopmental disorders, ADHD has an early onset in the lifetime, with manifestations typically occurring in early childhood or during formal education. Therefore, several symptoms of inattention and/or hyperactivity are to be present before age 12 for a child to be diagnosed with ADHD, according to Criterion B of the DSM-5. The choice for this age limit instead of an earlier age is due to difficulties in establishing the precise onset of symptoms retrospectively, since adult recall of childhood events tends to be inaccurate. It is worth noting, however, that the ICD-10 advocate for age 6 as a limit for the onset of the symptoms (World Health Organization, 2015); nonetheless, they make a caveat that only extreme levels of hyperactivity should lead to a diagnosis in preschool children, given the normal broad variability of motor activity in young children.

Besides this early onset, the symptoms must occur at least in two different settings of the child's life, for example at school and home, or with family members and friends (Criterion C). There is variability in the display of symptoms according to the context in a certain setting, so that they may be discreet or even absent in certain situations, for example when the child is being closely supervised, receiving frequent and contingent rewards for good behaviour, interacting in one-on-one situations (e.g. during a medical consultation), is engaged in an activity of his interest, has external stimulation (e.g. playing with electronic devices). To accurately assess the presence of symptoms across different settings, informants who have seen the child in those environments must be consulted. The symptoms must clearly interfere negatively in the child's quality of social, academic or occupational functioning (Criterion D) and they must not solely occur during the course of a psychotic disorder nor be better explained by another mental disorder (e.g. mood or anxiety disorders, personality disorders, substance intoxication or withdrawal, dissociative disorder, Criterion E).

As presented above, the symptoms in the DSM-5 are clustered in two different types: inattentive and hyperactive-impulsive symptoms. This is due to different types of ADHD manifestation: a child can either manifest predominantly inattentive symptoms (if Criterion A1 is met for the past six months, but not Criterion A2), or predominantly hyperactive/impulsive symptoms (if Criterion A2 is met on the past six months, but not Criterion A1), or a combined presentation of symptoms (if both Criterions A1 and A2 are met for the past six months). This classification of subtypes of the disorder, however, is not endorsed by the ICD-10. While DSM-5 specifies three subtypes of ADHD based on the division between attentional and hyperactive-impulsive symptoms, ICD-10 subdivides

the hyperkinetic disorders (disease code F90.-) into four further subtypes: Disturbance of activity and attention (code F90.0), Hyperkinetic conduct disorder (code F90.1), Other hyperkinetic disorders (code F90.8) and Hyperkinetic disorder, unspecified (code F90.9). As can be noted, the typology proposed by the ICD-10 rather emphasizes the division between conduct (i.e. one's compliance to social rules), and activity and attention (i.e. overall behavioural baseline and attentional involvement to tasks).

This divergence between the classifications reflects a debate in the medical literature about the existence of two separate disorders corresponding to the inattentive and hyperactive subtypes of ADHD. This debate is driven by the fact that, although the patterns of clinical presentation of both subtypes are clearly distinct, they are not always stable during the development, there is a large genetical overlap for the two groups of symptoms, and their purported different etiologies were not yet fully identified (Greven, Asherson, et al. 2011; Greven, Rijsdijk, & Plomin, 2011; Lahey et al., 2005; Larsson et al., 2006; Leopold et al. 2019; Kuntsi, Pinto, et al., 2014; Kuntsi, Wood, et al., 2010; Willcutt et al. 2000). As an instance of the controversy, large twin studies have shown that the genetic heritability for both hyperactive-impulsive and inattentive traits are about 70%, but the genetic correlation for hyperactivity-impulsivity and inattentiveness is 0.55, meaning that albeit the two dimensions of ADHD have overlapping genetic components, they are also largely controlled by specific genetic effects (Greven, Rijsdijk, & Plomin, 2011; Greven, Asherson, et al. 2011).

The DSM-5 tends to endorse the literature suggesting the existence of different disorders for each of the two dimensions of ADHD (inattention and hyperactive-impulsivity), whereas the ICD-10 deliberately exempts itself from doing so, by stating that knowledge on specific aetiology is lacking at present. Moreover, the ICD-10 clearly states its avoidance in using the term "attention deficit disorder" because the underlying psychological processes for such disturbance in attentional functioning are not solidly established (World Health Organization, 1992, page 262). Instead, the ICD-10 shows a more conservative approach by clustering hyperkinetic disorders with conduct disorders, thus maintaining the view of the trouble as akin to behavioural maladjustments rather than a reflection of underlying disruptions of the cognitive functioning.[11] We will emphasize the two-dimensional approach of ADHD (as proposed by the DSM-5) in the remaining of this

[11] This approach was maintained since the older versions of the manual and explicitly explained in the 1992 version, as cited. Recent versions of the manual (i.e., 2015, 2016) kept this organization.

thesis because it reflects a tendency in the contemporary literature and because it has great value for the understanding of children's cognitive functioning, and thus for the aims of our research.

Besides prescribing the specification of what dimension of ADHD a patient mostly manifests, the DSM-5 also prescribes the specification of the current severity of the symptoms, and if they are in partial remission. The severity of symptoms is classified in mild, moderate or severe depending on their impact on the child's functioning. If few or any symptoms exceeding the number required to make the diagnosis are present, and they cause only minor impairments in social or occupational functioning, the ADHD is considered mild. If many more symptoms than the amount required for diagnosis are present, and they cause marked impairments in the child's functioning, then ADHD is considered severe. If the functional impairment and the number of symptoms are between "mild" and "severe", then ADHD is considered moderate. The DSM-5 does not provide clinicians with further details on how to specify the severity of symptoms, so professionals heavily rely on their clinical experience to do so. Lastly, ADHD is considered in partial remission if full criteria were previously met but fewer than the number required has been met in the past 6 months, and the symptoms still cause impairments in social and occupational functioning.

While making a clinical judgement about the presence of symptoms and their severity, clinicians must consider the child's age, intelligence quotient and the situation where symptoms take place. The child's overactivity and deficits in attention should be diagnosed solely if they exceed the expected for that age and IQ. Some associated features support the diagnosis but are not necessary nor sufficient for it. Amongst them are low frustration tolerance, mood lability, irritability, disinhibition in social interactions, ignoring social rules (e.g. difficulty in waiting for turns, interrupting others), and low academic achievement despite the absence of a learning disability. Concerning the emotional features of ADHD, many children may develop low self-concept, depression, and secondary symptoms of anxiety related to school or work performance. These mood disorders can co-occur with ADHD during childhood and prolongate to adolescence and adult years, accompanying the individual during his lifespan (D'Agati et al., 2019; Grogan et al., 2018). The comorbidities and the differential diagnosis of ADHD will be further detailed in the next topic.

2.2. Comorbidities and differential diagnosis

Comorbidity is a common condition in children and adolescents with ADHD. In this section, we chose to report prevalence rates from studies published from 2012 onwards, which is the year of publication of the 5th edition of the DSM. Although some of these studies include data from patients diagnosed with criteria from DSM-IV or DSM-IV-TR (American Psychiatrical Association, 1994, 2000), they were carried out after the release of DSM-5, therefore we believe that they are more likely to reflect the current medical view of ADHD. Also, we excluded studies about comorbidities in the adult population and those whose main disorder was not ADHD (i.e. studies investigating the presence of ADHD as a secondary diagnosis).

The prevalence estimates of comorbidities in children diagnosed with ADHD range from 52% to 88.6% according to studies with European, Latin American, and Middle Eastern populations (Amiri et al., 2013; de la Barra et al., 2013; Jensen & Steinhauser, 2015; Reale et al., 2017; Zorlu et al., 2015). This inconsistency on prevalence rates is due mostly to differences in sample sizes, criteria for the selection of medical records, and temporal criteria adopted for the co-occurrence of disorders. Some authors, for example, adopt sequential comorbidity (when one disorder follows the other during the life span) for their estimates, whereas others adopt concurrent comorbidity (when two or more disorders happen at the same time) as a criterion.

Most of the studies indicate a prevalence rate of comorbidities between 60% and 70% (Amiri et al., 2013; Reale et al. 2017; Zorlu et al. 2015). The highest prevalence rate reported, 88%, comes from a Chilean sample of 1,558 children and adolescents aged 4-18 (de la Barra et al., 2013). It is worth noting, however, that this study used structured interviews with families outside a clinical setting and included participants without a formal diagnosis for a psychiatric disorder. The lowest prevalence rate for comorbidities reported, 52%, comes from a study including 14,825 patients aged 4-17 years, diagnosed in Danish psychiatric hospitals between the years of 1995 and 2010 (Jensen & Steinhausen, 2015).

The most common comorbidities for ADHD are conduct disorders, learning disorders and anxiety disorders. Again, there is no agreement in the literature about the prevalence rates for each one of these comorbidities, but conduct disorders appear to be the most frequent additional diagnoses to ADHD since they are the first one cited both by

the DSM-5 and ICD-10. A brief comment on the nomenclature about conduct disorders is necessary before continuing our exposition. In the DSM-5, the oppositional defiant disorder and conduct disorder are two different diagnostic categories, both appertaining to the class of "disruptive, impulse-control, and conduct-disorders" (American Psychiatric Association, 2013). In the ICD-10, the oppositional defiant disorder (disease code F91.3) belongs to the broader category of conduct disorders (disease code F91.-, World Health Organization, 1992). Epidemiological studies report the prevalence rates for comorbidities either separating conduct disorder from oppositional disorder, in accordance to the DSM-5 nomenclature, or by presenting them in the clustered category of "conduct disorders" (ICD-10 nomenclature). For the sake of brevity and because further exploring the clinical profile of patients with ADHD is out of the scope of this thesis, we will adopt the nomenclature "conduct disorders" henceforth in this section.

According to Jensen and Steinhausen (2015), conduct disorders are the main co-occurring diagnoses to ADHD (16.5% of the global sample), respectively followed by learning, language and motor developmental disorders (15.4%), autism (12.4%), and intellectual disability (7.9%). Their study included a big sample of participants (14,825 children and adolescents) who received a formal diagnosis in public hospitals during a long time frame (from 1995 to 2010). Other studies, however, indicate that learning disorders are the leading additional diagnoses to ADHD. In the study of Reale et al. (2017), they correspond to 56% of the cases where a patient received at least one supplementary diagnosis (66% of the global sample), followed by sleep disorders (23%), oppositional defiant disorder (20%) and anxiety disorders (12%). In the study of Zorlu et al. (2015), learning disorders correspond to 35.7% of the cases with at least one comorbidity (60% of the global sample), followed by conduct disorders (27.8%), and anxiety disorders (6.1%). We highlight the fact that the aforementioned percentage values for the studies of Reale et al. (2017) and Zorlu et al. (2015) are respective to the number of cases of ADHD who presented an additional diagnosis, whereas the values reported by Jensen and Steinhausen (2015) are respective to the global number of ADHD cases.

Concerning the process of differential diagnosis, the DSM-5 lists the following disorders, in this sequence: oppositional defiant disorder, intermittent explosive disorder, specific learning disorder, autism spectrum disorder, intellectual disability, reactive attachment disorder, anxiety disorder, depressive disorder, bipolar disorder, disruptive mood regulation disorder, substance use disorder. It is noticeable that disorders related to

conduct adjustments and to learning difficulties have preponderance upon other disorders during an assessment for differential diagnosis. This is due both because they share many symptomatic similarities to ADHD and because they are the most frequent comorbidities in children and adolescents who have received an ADHD diagnosis, as exposed above. We will not address the clinical assessment and treatment of ADHD in this chapter as they are out of the scope of the thesis.

In the previous section, we introduced the scientific debate about the two dimensions of ADHD and a proposal of considering them as two separate disorders. A relevant matter for this discussion is the fact that the presentation of comorbid disorders varies depending on whether the patient exhibits an inattentive or hyperactive-impulsive profile. In addition, the type of comorbidities presented by patients is sensitive to sex, which is also a predictor for the subtype of ADHD diagnosis (Arnett et al., 2015; Mowlem et al., 2019; Ottosen et al., 2019). In the following section, we will further explore the discussion about the three subtypes of ADHD (inattentive, hyperactive-impulsive, and combined presentation) by exposing particularities regarding their distribution in the population according to sex and age, their most frequent comorbidities, and their onsets during the lifespan. These particularities shall be integrated to provide a more comprehensive view of the differences between the two main subtypes of ADHD.

2.3. The different subtypes of ADHD

We stressed above that the DSM-5 divides the symptomatology of ADHD in two main dimensions and that there are three possible patterns of clinical presentation: predominantly inattentive, predominantly hyperactive/impulsive, and combined presentation. It is well documented in the literature that ADHD diagnosis is more prevalent in boys than in girls (male to female ratio is about 3:1, Willcutt, 2012) and that the clinical subtypes are not equally distributed between the sexes. Boys are less likely than girls to meet criteria for the inattentive type (Yüce et al. 2013), despite being the majority of cases in all subtypes of ADHD (male to female ratio above 1 across all age groups, Willcutt, 2012), and presenting more severe scores than girls in separate measures of both inattention and hyperactive-impulsivity (Arnett et al. 2015). However, some authors consider that ADHD is actually misreported and misdiagnosed in girls. Girls' symptoms tend to be overlooked by adults because their externalizing behaviours are less prominent and they tend to develop better coping strategies than their male peers (thus better

academic achievement) – what causes girls to be less referred to medical assessment, hence to diagnosis and pharmacological treatment (Mowlem et al., 2019; Quinn & Madhoo, 2014; Rucklidge, 2010).

The subtypes of ADHD not only reveal sex differences in the population, but also differences in the patterns of comorbidities: conduct disorders are more common in the hyperactive-impulsive and combined subtypes (Elia et al., 2008; Huh et al., 2011; Yüce et al., 2013), whereas learning disorders are more common in the inattentive subtype (Baeyens et al., 2006; Zorlu et al, 2015). The type of comorbidity also interacts with sex, with boys under higher risk of having comorbid conduct disorders, and girls of having comorbid anxiety disorders (Jensen & Steinhausen, 2015; Kraut et al. 2013; Yüce et al., 2013). This is aligned with what was previously said about externalizing symptoms being more common in boys than in girls (but see also Ottosen et al., 2019 and Zorlu et al., 2015, who present alternative results).

Regarding the onset and course of the disorder, ADHD is usually identified and diagnosed during elementary school, with the majority of diagnoses occurring between ages 6 to 10 depending on the sample (Danielson et al., 2018; Holden et al., 2013; Jensen & Steinhausen, 2015; Visser et al. 2014). Boys are significantly younger than their female peers at the moment of the diagnosis (Jensen & Steinhausen, 2015), which may reflect the misreporting and misdiagnosing scenario mentioned above. Nonetheless, the onset of the disorder precedes the entrance in formal education, with hyperactive-impulsive symptoms manifesting already in preschool children (DuPaul et al., 2001, Leblanc et al. 2008). A meta-analysis conducted by Willcutt (2012) including 97 studies with children, adolescents, and adults showed that the hyperactive-impulsive subtype is more frequent in preschoolers (52% of the cases) and gradually declines during elementary school-age (26% of cases) and adolescence (14%). Conversely, the prevalence of the inattentive subtype increases from preschool (23% of cases) to elementary school (45% of cases) and remain high in adolescence (72%); it is also the most frequent ADHD subtype in the adult population (47% of all adult cases). Lastly, the frequency distribution of the combined subtype slightly increases from preschool (25% of cases) to elementary school (29% of cases), and then declines during adolescence and adulthood (14%).

This distribution pattern of the subtypes across ages suggests that there is a shift in the symptoms during the transition from preschool to elementary school. The increasing

demands of attention make the inattentive symptoms more prominent and more impairing, which causes prevalence rates of the combined subtype to increase, and rates of the hyperactive-impulsive subtype to decrease in this age group. Concurrently, hyperactive-impulsive symptoms decline with age whereas inattentive symptoms are stable during the development, causing possible changes in diagnoses: from elementary school onwards, some children who have met criteria for ADHD-combined subtype during preschool age may shift to the inattentive subtype (Willcutt, 2012). In summary, we can outline two main pathways for ADHD development during the life course: 1) hyperactivity-impulsive symptoms arising in early childhood and then being gradually succeeded and/or replaced by inattentive symptoms during elementary-school-age and adolescence, and 2) the co-occurrence of hyperactive-impulsive and inattentive symptoms in early childhood is succeeded by a reduction in the levels of hyperactivity-impulsivity as inattention remains stable. These developmental trajectories will be resumed later in the thesis and must be bearded in mind for the understanding of our rationale.

2.4. Dysfunction of some core cognitive processes in ADHD

We exposed in previous sections that children with ADHD have high rates of comorbid learning disabilities and commonly face difficulties during formal education. They have lower scholastic achievement when compared to their typically developing peers, have more reading disabilities and/or perform poorer in reading comprehension tasks, and face substantial difficulties in mathematical cognition (Cain & Bignell, 2014; DuPaul et al. 2013; Frazier et al. 2007; Miller et al., 2013; Tosto et al, 2015; Wadsworth et al., 2015). A meta-analysis conducted by Frazier and colleagues (2007) including 72 studies showed medium to large effect sizes of ADHD upon overall school achievement (Cohen's d = 0.71) and upon the three main achievement areas: reading (d = 0.73), mathematics (d = 0.67), and spelling (d = 0.55). These associations between ADHD and learning difficulties are stronger amongst children with the inattentive profile (Massetti et al., 2008; Tosto et al., 2015), and the genetic association between inattention and reading difficulties is detectable from kindergarten, as shown by a large multi-cultural twin study (Ebejer et al., 2010).

The commonality between the academic abilities aforementioned is that they are all reliant in executive functions (Diamond, 2013), thus they rather reflect impairments in basic cognitive processes than in the ability to perform specific tasks. Their association

with inattention from an early age suggests a constitutional interplay between basic cognitive processes, academic skills, and the emergency of ADHD symptoms during the development. Indeed, studies suggest deficits in executive functions (specifically working memory and response inhibition), alongside with delay aversion, as potential endophenotypes of ADHD (Bitsakou et al., 2009; Castellanos & Tannock, 2002; Crosbie et al., 2013; Doyle et al., 2005; Gau & Shang, 2010; Tarle et al., 2017; Willcutt et al., 2005).

Endophenotypes are quantitative internal traits, either cognitive or physiological, that reliably reflect the functioning of a neural system associated with a given pathology. They are heritable traits that segregate with illness in the population (e.g. people affected by a certain pathology will exhibit higher levels of its associated endophenotypes) and co-segregate with illness in families (e.g. a non-affected sibling must not present an associated endophenotype as well), thus being a useful construct to identify the genetic variants causing a disease (Gottesman & Gould, 2003). Endophenotypes are assumed to be more closely related to the organic causes of disease than its behavioural phenotypes (i.e. the clinical presentation of symptoms), for instance: deficits in executive functions have a more direct link with dysfunctions in the frontoparietal network than hyperactive behaviour *per se*. Therefore, endophenotypes are regarded as the bridge between low-level genetic determinants of a disease and its broader high-level symptoms.

In the case of ADHD, evidence from both behavioural and neurofunctional studies indicates that deficits in executive functions (low working memory capacity and poor inhibitory control) and delay aversion are plausible endophenotypes. At the behavioural level, children's executive functions are assessed by computerized experimental measures (e.g. reaction times, proportion of correct responses, error rates) or composite scores from standardized tests (e.g. intelligence measures such as verbal IQ). Delay aversion, which is an index of motivational functioning, in its turn, is measured by a child's choices in tasks involving immediate or postponed rewards. At the neurofunctional level, data from neuroimaging studies reinforce behavioural evidence by revealing altered brain networks involved in those basic psychological processes. Two meta-analyses of functional magnetic resonance imaging (fMRI) studies showed the hypoactivation of the right inferior frontal cortex, supplementary motor area, anterior cingulate area (which is considered as supporting inhibition), hypoactivation of the dorsolateral prefrontal cortex, frontoparietal and ventral networks (considered as the attentional network), and the hyperactivation of the default and somatomotor networks (which are related to overall control of baseline

motor activity during rest) (Cortese et al., 2012; Hart et al., 2013). Accordingly, O'Halloran and colleagues (2018) found that abnormally strong connectivity in motor networks are related to impairments of sustained attention in adolescents with ADHD and typically developing adolescents.

We will now outline data about the three main endophenotypes of ADHD in order to compose a general portrait of those children's cognitive profile and motivation. We will start by delay aversion and then proceed to the deficits in inhibitory control and working memory. We will focus on the latter since it is the theme under investigation in the present thesis.

2.4.1. Delay aversion

Delay aversion is the behavioural tendency of choosing smaller immediate rewards over larger delayed rewards; in other words, it is the motivational tendency to avoid delays. Children with ADHD consistently demonstrate a preference for small immediate rewards in experimental choice paradigms when compared to typically developing children. This preference occurs in conditions of both pre- and post-reward delays (Sonuga-Barke et al., 2008). Interestingly, their delay aversion is greater in the absence of post-reward delays, and when overall delay can be avoided as a result of their choices. Together, these response patterns in choice-tasks demonstrate that ADHD children not only have low sensitivity to delayed rewards but also an overall aversion to delayed events during a task, irrespective to the presence and/or magnitude of the reward. Accordingly, adolescents with ADHD have greater activation in the amygdala – a brain area known for being involved in processing aversive events – when an upcoming delay interval is cued during a choice task (Van Dessel et al., 2018).

The depreciation of reward value over time is not an exclusivity of the motivational functioning of children with ADHD. This is a common phenomenon in organisms and it is an important element of decision-making (Da Matta et al., 2012). It is referred to as delay discounting in the literature, and the function between the subjective value of the reward and time is used to estimate one's tendency to devaluate rewards. The steeper is a delay discounting function, the more rapidly the organism "discounts" (i.e., depreciates) the value of that reward. Functions of delay discounting in experimental tasks are an index of one's impulsivity and self-control, as they reflect a subject's behavioural tendency in making optimal or sub-optimal choices in terms of reward amount. High rates of delay

discounting express impulsive choices (i.e., choosing a small immediate reward when a greater reward would be available later), whereas low rates of delay discounting express self-control (i.e. postponing an immediate reward to receive a more advantageous one, albeit delayed).

Delay discounting functions are significantly steeper for ADHD children between ages 7 and 9 when compared to controls, but this effect disappeared when controlling for IQ in a study by Wilson and colleagues (2011). Steeper delay discounting functions are also associated with risk-taking behaviour, impulsivity and sensation-seeking in adolescents (Baumann & Odom, 2012; Romer et al., 2010). However, sensation-seeking was not related to delay discounting in a sample of children at high risk of developing ADHD (Burns et al., 2020).

2.4.2. Response inhibition

Response inhibition is the executive function responsible for withholding behavioural responses. It is an essential cognitive ability to refrain preponderant impulsive responses, therefore, to achieve goal-adjusted behaviour. In experimental settings, response inhibition is commonly assessed by stop-signal-tasks (also known as Go/No-go tasks) and Stroop tasks. In the stop-signal-task, participants must respond differently to two classes of stimuli, by making a response to a signal (the "go" signal) and not responding to another (the "stop" signal). Because the release of a stop signal is unpredictable and preceded by a series of go signals, the underlying psychological process is the inhibition of a motor response that was about to be initiated – in other words, an ongoing action motor plan. In the classic version of the Stroop task, participants must respond to the ink colour of written words (colour names, either matching or mismatching the ink colour) and say it aloud, but without reading the word. In cases of mismatches between the ink colour and the written colour name, participants exhibit slower response times and are more prone to errors, which demonstrate a failure in inhibiting an interfering automatic process (reading). In the clinical setting, computerized continuous performance tests (CPT) are used to measure selective attention, sustained attention, and impulsivity to support the diagnostic process of ADHD (Berger et al., 2017; Hall et al., 2016). The CTP task consists of a rapid sequential presentation of visual (usually letters, digits, or geometric figures) or auditory stimuli over a prolonged period of time. Participants must respond to a target stimulus and avoid responding to non-target stimuli, that is, to inhibit a response.

The variables of interest in tests of response inhibition are commission errors (responding when a response should be avoided), omission errors (not responding when a response was expected), and response times (RTs). Commission errors denote failure in inhibiting a preponderant response and are regarded as in index of impulsivity, and omission errors denote misses in detecting task-relevant stimuli and are assumed to measure sustained attention; the mean RT and RTs variability, in their turn, are indexes of processing speed and reflect the duration of the decision-making process.

Classically, children with ADHD have more commission and omission errors and show greater RTs variability than typically developing children (Berger et al., 2017; Lijffijt et al., 2005). This greater RTs variability is attributed to a larger number of rapid responses and slow responses in comparison to the average RT performance of typically developing children. Short RTs associated with commission errors reveal impulsive responses, whereas slow RTs associated with commission and omission errors reveal inattention. Children with ADHD make significantly more of both types of errors than typically developing children in stop-signal tasks, as revealed by Berger et al. (2017) in a study with 798 participants.

Lijffijt and colleagues (2005) conducted a meta-analysis with 29 studies comparing RTs of ADHD patients and controls in stop-signal tasks. The effect sizes (Cohen's d) of ADHD across all studies (both children and adults included) was d = 0.29 for the mean RT, d = 0.65 for the within-subject standard deviation of RT, and d = 0.58 for the stop-signal RT (i.e., RTs only in "stop" trials). These results indicate that RTs in stop-signal tasks remain as a reliable index of impulsivity and inattention in ADHD throughout the development, yet there are some particularities concerning children and adults' performance. The presence of an ADHD diagnosis yielded no differences in the mean RT for adults (pooled d = - 0.02), whereas it caused a moderate effect in the mean RT for children (pooled d = 0.52). In addition, the effect of ADHD upon within-subject variability was greater for children (d = 0.72) than for adults (d = 0.44); on the other hand, the effect upon stop-signal RT was greater for adults (d = 0.79) than for children (d = 0.58). Taken together, these results show three phenomena: 1) adults with ADHD tend to respond similarly to controls in terms of mean RT (i.e. no difference between the overall RTs), 2) the distribution of individual RTs is more heterogeneous in ADHD children than in ADHD adults (greater inter-individual variability of response latencies attributed to the presence of ADHD), and 3) latencies in stop-signal trials tend to elongate in adulthood,

suggesting the permanence of impulsive responses despite a more time-consuming decision-making process (i.e. more "watchful" commission errors).

Albeit evidence on ADHD children's impaired performance in stop-signal and CTP tasks, an important caveat must be noticed about some results with Stroop tasks. On the one hand, studies using Stroop tasks present behavioural and neurofunctional results aligned to those presented above (Peterson et al., 2009; Ikeda et al., 2013; Kóbor et al., 2015). On the other hand, a meta-analysis of 25 studies using the classical Stroop colour-word test (Schwartz & Verhaegen, 2008) found that Stroop interference is not larger in ADHD individuals than in age-matched controls and that there is no evidence of differential maturational effects on performance in the Stroop task, regardless of ADHD diagnosis. Although ADHD individuals are on average 1.14 times slower than typically developing participants, the slope of the RT function was identical across age groups and between clinical vs. control groups. The authors suggest that maturational rate in performance is the same for individuals with and without ADHD and that the Stroop effect is immune to age in both cases. To conclude, there is conformity among studies using different response inhibition paradigms about the fact that individuals with ADHD exhibit higher levels of impulsivity and inattention, as attested by higher rate errors and greater RTs variability. Nonetheless, findings diverge about the existence of maturational effects detected by RTs.

2.4.3. Working memory

In experimental settings, working memory capacity is assessed by performance in tasks involving either recognition or recollection of verbal and/or visuospatial material. A participant's performance is measured by his response accuracy (e.g., percent of correct responses, indexes of discrimination between targets and non-targets), reaction times, and memory span (i.e. the maximum number of items that one can recall). In their turn, standardized neuropsychological batteries commonly reproduce computerized experimental tasks with physical materials (e.g., paper and pencil, cubes) and fewer number of trials. They use composite scores relative to the performance of the general population instead of comparing raw measures of performance in different conditions. Studies about working memory functioning in children with ADHD use both the experimental and the neuropsychological testing approaches, and we will present them together henceforth.

The majority of studies in the literature confirm that performance in tests of working memory affects the academic achievement of children with ADHD, regardless of the outcome measured by researchers (individual grades, scores on math tests, reading tests, grade point average) (Fried et al., 2019). Working memory is accountable for most of the variance in reading comprehension in children with ADHD, even when reading ability is controlled (Miller et al., 2013). In the mathematics domain, accuracy in n-back tasks and parent-rated inattention predict the variance in maths achievement of ADHD children, which suggests that deficits in working memory and sustained attention account for their poorer achievement outcomes (Antonini et al., 2016). These results are in line with the mediation model proposed by Rogers and colleagues (2011), according to whom the achievement of children with ADHD in reading and mathematics is predicted by behavioural inattention and performance in phonological and visuospatial working memory. In their model, phonological working memory was associated with achievement both in reading and mathematics, whereas visuospatial working memory was associated only to math achievement.

When the components of WM are considered separately, the presence of ADHD is more deleterious to performance in the visuospatial domain than in the phonological domain, as demonstrated by larger effect sizes both for experimental measures and standardized neuropsychological tests (Martinussen et al., 2005; Kasper et al., 2012; Willcutt et al., 2005). The meta-analyses conducted by Martinussen and colleagues (2005) and by Willcutt and colleagues (2005) showed effect sizes (Cohen's d) ranging from 0.63 to 1.06 for visuospatial working memory measures and from 0.43 to 0.55 for phonological working memory measures, depending on the moderators included on the analyses. Kasper and colleagues (2012) found an effect size (Hedge's g) of 0.74 for visuospatial working memory and 0.69 for phonological working memory. The Cohen's d and the Hedge's g are relatable measures of effect sizes for sample sizes larger than 20, which yields reliable comparisons between the meta-analyses' results (Durlak, 2009). As can be noted, ADHD has medium to large effect sizes upon performance in visuospatial working memory tasks, whereas it has small to medium effect sizes in phonological tasks. Jointly, these studies demonstrate that impairments in the visuospatial domain are accountable for most of the differences between the performance of ADHD and typically developing children.

Besides controlling for the working memory domain involved in the tasks, Martinussen and colleagues (2005) controlled the type of requirements in working memory operations, that is, they separated the studies according to the involvement of memory storage and central executive demands. Tasks with central executive requirements yielded larger effect sizes than tasks with simply storage in the visuospatial domain, but not in the verbal domain. In the visuospatial domain, the effect size of ADHD upon performance was d = 0.85 for storage and d = 1.06 for central executive; in the verbal domain, the effect size was d = 0.47 for storage and d = 0.43 for central executive.

Finally, a study by Kofler and colleagues (2018) addressed the role of the episodic buffer in working memory deficits of children with ADHD, by asking them to perform a multimodal-binding memory task in addition to visuospatial and phonological tasks. In the visuospatial task, participants should reproduce spatial sequences containing black dots and a single red dot in a grid, assigning the position of the red dot at the end of the trial. In the phonological task, they performed a letter-digit sequencing task, in which they should recall the digits in ascending order and recall the letter at the end of the trial. In the task designed to tap the episodic buffer, participants performed a letter-digit sequencing task in which the stimuli were visually presented in a spatial grid. They were required to assign the locations of the digits in ascending order and to assign the location of the letter at the end of the trial. For example: in a trial with the sequence 6-2-M-5, the correct response should be to point to the locations previously occupied by 2, 5, 6, and M in this order. Note that all the three tasks (visuospatial, phonological, and episodic buffer) involved both storage and central executive demands. The authors found that ADHD children performed significantly worse than controls in all three working memory tasks, and that the episodic buffer task decreased accuracy for both groups. However, the addition of an episodic buffer demand did not differentially affect the ADHD group. Rather, they proposed that poorer performance in ADHD is more parsimoniously explained by an overall working memory deficit (probably linked to central executive impairments) than by a specific impairment in the episodic buffer (but see also Alderson et al., 2021 for an alternative view).

To summarize, data show that ADHD children have impairments in all domains of working memory and that these impairments are more pronounced in the visuospatial domain and in operations requiring the involvement of the central executive. Together with data about deficits in sustained attention (Huang-Pollock et al., 2012; O'Halloran et al., 2018; Tucha et al., 2017) and poorer performance in children with the inattentive type

in broader academic domains (Massetti et al., 2008; Tosto et al., 2015), these data might suggest that the typically observed working memory weaknesses in ADHD reflect a central executive deficit and a malfunction of attentional refreshing. This is a central point for this thesis, and it will be resumed later. To finish our presentation of ADHD, we will expose the main theoretical models of ADHD functioning.

2.5. Theoretical models of ADHD functioning

The first scientific description of ADHD was made by Still in 1902 when he presented a syndrome characterized by a "morbid defect of moral control". In the following decades, the vision that ADHD was caused by a defect of moral behaviour shifted to the notion that it was rather caused by a minimal brain injury, mostly because of reports on behaviour disorders after encephalitis lethargica, and studies on birth trauma and its relations with mental impairments in children. Later, Kramer and Pollnow (1931, cited by Lange, 2010) described what they called "hyperkinetic disease of infancy", with an emphasis on hyperactive motor behaviour and symptoms of restlessness. In the 1950s, the use of stimulant medication showed a remarkable effect on misbehaved children, especially upon their inattentive symptoms, which made the medical community to consider them as part of the disorder. The acceptance of inattention as a core characteristic of ADHD would only occur during the 1970s, after a paper by Douglas (1972) advocating for deficits in attentional control and impulse inhibition as more compelling central features of ADHD than hyperactivity itself. It was only in 1980, in the third edition of the DSM (DSM-III, American Psychological Association, 1980) that ADHD was characterized as a disturbance of both attention and hyperactivity (see Lange et al. 2010 for a comprehensive historical view of the literature on ADHD).

As exposed in the previous sections, the current vision of ADHD considers both groups of symptoms and, in terms of etiological explanations, focuses on basic psychological processes rather than organic malfunctions. The contemporary theories about ADHD functioning are grounded in evidence from the cognitive profile of patients in experimental tasks, standardized tests, and neuroimaging studies. All theories acknowledge that ADHD is a behavioural disturbance caused by central dysfunctions, but they essentially diverge on which central process is the source of the cognitive deficits and related peripheral symptoms. In the following topics, we will outline the main contemporary theoretical models of ADHD, each of them primarily considering one basic

psychological process as the core cause of the disorder: behavioural inhibition, motivation, and working memory.

2.5.1. Barkley's (1997) inhibitory model of ADHD

One of the most prominent models of ADHD was proposed by Barkley (1997) and mainly focuses on children with the hyperactive-impulsive profile. His model does not comprise the set of symptoms of the predominantly inattentive type of ADHD. This particular theoretical choice is aligned with Barkley's position in the debate about the separation of ADHD in two broader disorders that was exposed in section 2.1. The author has defended since the 1990s (Barkley et al., 1990) that the predominantly inattentive type is not a subtype of ADHD, but rather a different disorder (an in-depth discussion on this issue can be found in Barkley, 2001 and Milich et al., 2001).

The central postulate of Barkley's model is that the essential impairment of ADHD is a deficit involving response inhibition, which leads to secondary impairments in four neuropsychological abilities that partially depend on inhibition: working memory, self-regulation of affect-motivation-arousal, internalization of speech, and reconstitution. He conceptualizes behavioural inhibition as three interrelated processes, that act together to adjust goal-directed behaviour: a) inhibition of the initial prepotent response to an event (i.e. the impulsive response); b) stopping of an ongoing response, which allows a delay in decision-making; c) the protection of this delay period and the self-directed responses that occur within it (e.g. reasoning) from interference and/or disruption by competing events or responses (i.e. interference control). A failure in one of these three processes of response inhibition leads to failure in achieving the effective execution of the four neuropsychological abilities stated in his model, henceforth called "executive functions" in conformity to Barkley's terminology. At the end of the chain, impairments in the executive functions will cause disturbances in motor control, fluency, and syntax, resulting in the behavioural symptoms of ADHD. We will now outline Barkley's propositions on how a deficit in response inhibition affects the four executive functions stated in his model, in the same order that he presented in his paper (Barkley, 1997).

Working Memory. According to this model, children with ADHD are less controlled by internally represented information than their typically developing peers. To be "controlled by internally represented information" means that one can use information stored in memory systems to guide and adapt behaviour according to environmental

exigencies such as task requirements and social rules. Because working memory stores, manipulates, and updates information available to consciousness in a given time-point, impairments in working memory, result in difficulties in rule-governed behaviour, reasoning, and future planning. This causes behaviour of children with ADHD to be more controlled by the temporal "now" than by internal representations of the past (learning experiences), the future (ability to anticipate outcomes), and the sense of time (duration of activities). Indeed, at the behavioural level, children with ADHD exhibit all the difficulties predicted by the working memory module in Barkley's model as attested by studies on delay aversion and academic difficulties stressed above. However, the author does not provide a detailed explanation of the causal links between response inhibition and working memory, but rather limits himself to cite evidence on correlations between these two impaired psychological processes. Barkley acknowledges the lack of evidence in the predicted behavioural outcomes of the working memory module, by stating that little research had been conducted to specifically test those predictions. Through his further formulations on the functioning of response inhibition and other modules of the model, it is possible to apprehend that the link between response inhibition and working memory relies on the temporal suspension of actions promoted by the former. According to his model, working memory operations can only take place in the mind if impulsive responses are postponed.

Self-regulation of affect/motivation/arousal. This module of Barkley's model accounts for ADHD children's difficulties in emotional self-control, objective/social perspective-taking, self-regulation of motivation and arousal in service of goal-directed behaviour. His model predicts that ADHD children will be more reactive to emotionally charged immediate events (this characteristic sums with susceptibility to immediate environmental control, as explored above), will have fewer anticipatory emotional reactions to future emotionally charged events (as a result of the decreased capacity of forethought), have decrease capacity to deal with the impact of their own emotions on others, will have lessen capacities to self-induce and regulate motivational and arousal states, and will show greater dependence on external sources to maintain persistent effort in goal-directed behaviour. The two latter points derive from the fact that further away in time is an action's goal, the greater is the difficulty to sustain arousal and drive towards the goal. This point of the model is supported by evidence on ADHD children's delay discounting curves.

Internalization of speech. Uninhibited behaviour is associated to less mature self-directed speech, moral reasoning, and rule-governed behaviour both in typical developing and ADHD children (Kochanska et al., 1994; Zelato et al., 1995; Berk & Potts, 1991; Rosembaum & Baker, 1984, as cited by Barkley, 1997). A delay in maturation of internalization of speech results in impaired descriptions and reflections about events/environment, poorer abilities of problem-solving and self-questioning (since the internal speech organizes reasoning), difficulties in generating rules, thus affecting moral reasoning. Here again, the causal linkage between behavioural inhibition and internalization of speech is not clearly detailed by Barkley, but putatively as assumed to be a product of temporal delay in the action flow allowing verbal reasoning to take place.

Reconstitution. In Barkley's model, reconstitution refers to one's ability to spontaneously generate novel responses or ideas. Activities requiring a child to be creative, either in terms of motor response (e.g. novel motor sequence) or verbal discourse (e.g. telling a story, writing, interact with peers) reflect the operations of reconstitution. Uninhibited behaviour will prevent a child from executing reconstitution operations such as analysis and synthesis of behaviour, creativity in goal-directed behaviour, behavioural simulations (i.e. imagine the execution of certain response). This module of the model stands on evidence that children with ADHD exhibit poorer verbal fluency and discourse organization (Grodzinsky & Diamond, 1992; Tannock, 1996, as cited by Barkley, 1997).

Regarding the role of inattention in the model, Barkley considers it as a secondary symptom that stems from impairments in self-regulation on executive control of own's behaviour. He distinguishes two forms of sustained attention, that he calls persistence: one that is contingency-shaped and other that is self-regulated and goal-directed. The former is mostly a function immediate context (e.g. task novelty, reinforcement schedule), whereas the latter is an emergent property of the executive system, therefore self-driven and independent from immediate rewards. According to the author, self-regulated persistence is impaired in ADHD children and is probably different to the type of inattention seen in children with the predominantly inattentive type, who likely have impairments in focused/selective attention that are unrelated to self-regulation.

To conclude, Barkley's model proposes that poor inhibitory control accounts for all core symptoms of ADHD because it is a prerequisite for four essential executive functions (working memory, self-regulation of affect/arousal/motivation, internalization of speech,

reconstitution), which in turn modulate motor control, fluency, and syntax of motor responses, causing the behavioural display of symptoms.

2.5.2. The dual-path model of ADHD

The dual-path model of ADHD (Sonuga-Barke, 2005, Sonuga-Barke et al., 2008) is greatly influenced by Barkley's inhibition model. It has expanded the inhibition model by including a module accounting for influences in motivational states of the individual, besides the already mentioned intrinsic delay aversion of ADHD children. For this reason, it is also regarded as a motivational model of ADHD, in a sense that it casts light in social interactional variables acting upon motivation. Because this thesis will not address the impact of environmental variables such as reinforcement schedules or parental style upon symptoms of ADHD, we will not provide an in-depth discussion of this model. Instead, we will only highlight its additional theoretical propositions respective to Barkley's model.

The common ground for both models is that abnormalities in the reward brain circuitry and poor inhibitory control cause delay aversion (giving rise to impulsive behaviours) and impaired performance on executive tasks. The abnormalities in reward circuitry are a central module of Sonuga-Barke's model, with the status of primary causal variable alongside impairments in behavioural inhibition. This reward circuitry involves dopaminergic modulation in the basal ganglia-thalamocortical network (loop connecting the pre-frontal cortex to the striatum and caudate nucleus via excitatory glutaminergic cells), that is acknowledged as the neural substrate of inhibitory control (Aron, Monsell, et al., 2004; Aron, Robbins, et al., 2004; Eagle and Robbins, 2003). Besides, the neurobiology of impaired signaling of delayed rewards shares common elements with the basal-ganglia-thalamocortical loop (McClure et al., 2004). Based on this neurobiological evidence, Sonuga-Barke proposes that abnormalities in this circuitry cause low signals to future rewards and overall impairments in inhibition. Together, these two effects result in failures in waiting and poor performance in tasks, that usually are punished by the environment (e.g., adults, peers). Punitive responses to failed waiting and task failure modulate the system, culminating in generalized aversion to contexts of task execution and delayed contingencies (e.g., do the homework, sit in a classroom). According to this account, the symptoms of disengagement, lack of persistence, and hyperactivity are ways to avoid and/or escape punitive contingencies. In short, Sonuga-Barke's model considers that an intrinsic malfunction of inhibition and motivation networks adds to environmental

punishment to result in a behavioural pattern of avoidance and/or escape responses to aversive contexts.

2.5.3. The working memory model of ADHD

The model proposed by Rapport et al. (Rapport, Chung, et al., 2001; Rapport, Kofler et al., 2008; Alderson et al., 2010) is also grounded on evidence about impairments in executive functioning but offers an alternative view to the one proposed by Barkley (1997). This model is primarily concerned in correcting a major conceptual problem in the inhibition model, that is the attribution of causal dependence of working memory and other executive functions on the ability to inhibit impulsive responses. In Barkley's model, inhibition is regarded as a precedent event in the chain of mental processes during the execution of tasks, without considering that inhibition actually follows the registration of environmental events that prompts a prepotent response. An uninhibited response is a response to an environmental event. Thus, response inhibition is an intermediate element in the chain sensory register – inhibition – motor response. According to Rapport and colleagues, this raises the question of how sensory registration can directly elicit response inhibition without a mediator between these two processes. This bridging mechanism is ascribed to working memory, considered to have a primary controlling influence upon inhibition. Note that Rapport and colleagues' model inverts the causal relation between behavioural inhibition and working memory proposed by Barkley (1997). According to the authors, it is more parsimonious to consider behavioural inhibition as a product of working memory operations instead of a cause thereof.

From a theoretical standpoint, the authors argue that if working memory is conceived as the cognitive system that 1) organizes, interprets and maintain input stimuli; 2) retrieve long-term memory traces to interpret reality; 3) access and maintain mental representations of appropriate behaviour in response to input stimuli, then a failure in any of these operations should lead to random, tangential, or disorganized responses to environmental stimuli (Rapport et al., 2001). Their argument is based on evidence from bootstrap analyses of experimental data from stop-signal (response inhibition), spatial dots sequences (visuospatial working memory), and letter-digit sequence (phonological working memory) tasks, in which the central executive component in working memory tasks proved to be more a powerful estimate of overall performance than reaction times in the stop-signal task (Alderson et al., 2010). In their analyses, they created separate scores for each

component in each task: central executive, phonological storage, visuospatial storage, phonological composite, visuospatial composite, stop-signal reaction time, and stop-signal delay. The phonological and visuospatial composites corresponded to weighted measures of storage components and central executive demands in verbal and visuospatial tasks, respectively. The stop-signal delay corresponded to the adjusted delays between go- and stop- signals according to participant's performance during the task. Besides being the most powerful estimate of overall performance, the central executive component yielded the highest intercorrelations with all other components, followed by the visuospatial composite and the phonological composite.

Alderson and colleagues' (2010) results are in accordance with the meta-analyses presented in the previous section of this chapter (section 2.3) and stress the connection between central executive working memory processes and ADHD. The model originally proposed by Rapport and colleagues (2001) provides a detailed account of the relationship between central executive deficits and ADHD symptoms. In their model, failure in working memory leads to poor behavioural inhibition because retrieval from a long-term memory, short-term maintenance and processing (in the conscious focus of attention) of mental representations are disturbed, thus affecting the interpretation of immediate context and the planning and execution of adequate responses to environmental stimuli. Specifically, the authors consider decay as a core variable in their model and propose that rapid decay of information is responsible for working memory deficits.

Besides being considered the cause of disorganized behavior, the working memory deficit also drives children to redirect attention to other environmental stimuli and promotes high levels of motor activity (stimulation seeking). Note that the model conceptualizes attention as a targeting mechanism of the contents available in working memory at a given moment. The consequence of stimulation seeking is that the input rates of information in working memory increases, counteracting the effect of rapid fading of its contents, and promoting escape from a monotonous activity. The term "monotonous" is defined by the authors as the property of producing low rates of working memory activity, that may be derived either from low rates of stimulus input or perceptual constancy (i.e. minimal variation in sensory properties of the stimuli. Behavioral disinhibition is a direct consequence of rapid decay of working memory representations, whereas inattention and stimulation seeking are counteracting symptoms.

To summarize Rapport et colleagues' model, impaired working memory processes (rapid fading of memory representations) cause poor behavioural disorganization and stimulation seeking (as a way to compensate rapid fading) as primary psychological manifestations. These, in turn, cause poor performance in the cognitive (e.g. the aforementioned experimental tasks, academic tests) and behavioural (e.g. poor social judgments, inadequacy to contexts, conflict with peers) domains.

Through the literature review exposed in this chapter, working memory proved to be closely tangled to the psychological causes of ADHD, and likely to have a causal influence in the origin(s) of the symptoms. Noticeably, the working memory model of ADHD provides good accommodation to the results presented in the many fronts of research about ADHD showing that the disorder clusters with related impairments in executive functioning, including epidemiological, genetic, neurofunctional and experimental evidence. In the first chapter, we presented a working memory model – namely the TBRS model – that postulates two essential maintenance mechanisms, one of them relying on central executive attentional mechanisms (attentional refreshing), and the other in verbal-acoustic features of the stimuli (phonological rehearsal) (Barrouillet & Camos, 2012, 2021; Camos, 2015, 2017; Camos & Barrouillet, 2016, 2018). The relationship between ADHD and central executive deficits in working memory, as extensively evidenced above and formally theorized by Rapport and colleagues' model, is a valuable research route to investigate the two maintenance mechanisms depicted by the TBRS model and to validate them in a clinical population. In the next chapter, we will present a detailed explanation of the aims of this thesis, our hypotheses, and the specific predictions of the TBRS model for the performance of ADHD children in working memory tasks in comparison to their typically developing peers.

Chapter 3

The present thesis: Working memory deficits in ADHD

The theoretical knowledge on working memory and the clinical knowledge on ADHD are two research traditions that rarely intersect each other in the scientific literature[12]. In this thesis, we propose a convergent approach between the TBRS model and clinical research to study attention-based maintenance mechanisms in working memory. In Chapter 1, we have explained the agreement in the field that attention-based maintenance is crucial to visuospatial working memory. In Chapter 2, we have explained that children with ADHD have marked impairments of working memory functioning, in all domains and types of measurements, and that they are noticeably more impaired in the visuospatial domain. Also, we have explained that inattentive symptoms become increasingly more observable from elementary school-age to adolescence (Chapter 2, section 2.3), a period of optimization in attentional control, including the use of attentional refreshing (Gaillard et al., 2011) and the voluntary use of cues (Shimi et al., 2014) to boost memory representations (Chapter 1, section 2.2.4.2). These fundamental facts are at the origin of this thesis rationale.

3.1. Potential loci for an attentional deficit in working memory

Clinical literature and psychiatric reports unanimously assert that inattention is a pathognomonic sign of ADHD, and that impaired working memory performance is an associated trait of this disorder. At the behavioural level, the association between inattention and low working memory is revealed by research using the psychometric approach (i.e., symptoms scales and neuropsychological batteries). Nevertheless, the

[12] The work of Ortega et al. (2020) is an exception worth mentioning. In order to test if feature-binding in visuospatial working memory requires additional attentional resources, they compared a group of ADHD children to typically developing controls in a change detection task with a single-feature condition (only shape) and a bound-feature condition (color-shape binding). Memory for bound features was poorer than memory for a single-feature in both groups, with no significant interaction between group and condition. The authors interpreted these results as supporting the notion that binding operations in visuospatial working memory could be automated, outside the episodic buffer of the multicomponent model of working memory.

classical psychometric approach is not able to pinpoint specific psychological processes that underlie poorer working memory performance in ADHD. Experimental research must be called to action to offer a mechanistic explanation of this problem, namely *which* working memory mechanism(s) is(are) depleted in ADHD.

There are some potential loci to the attentional deficit in the working memory of individuals with ADHD. For instance, it has been suggested that children with ADHD have poor working memory because they cannot filter perceptual stimuli during encoding (i.e., impairment of selective attention, Jonkman, 2005; Jonkman et al., 2000, Jonkman et al., 2004), which overloads working memory with irrelevant mental representations. According to this view, the more information is encoded, the more mental representations are created and therefore it is more difficult to maintain information in working memory. Another account holds that individuals with ADHD cannot suppress competing items during retrieval in working memory tasks (Pollack et al., 2007, Storm & White, 2010), which could reflect difficulties in inhibition instead of attention *per se*. Finally – and more importantly for the goals of this thesis – working memory deficits could reflect difficulties in using attention to maintain items due to slow processing speed (Narimoto et al., 2018; Weigard & Huang-Pollock, 2017).

Narimoto et al. (2018) compared typically developing and ADHD children (10-year olds) in a spatial span task in which the presentation speed of the items and the retention interval was manipulated. The task had two experimental factors, presentation speed (fast vs. slow) and retention interval (immediate recall vs. delayed recall), orthogonally manipulated through four conditions. Their reasoning was as follows: If working memory deficits in ADHD reflect encoding limitations, then the slow presentation speed should benefit the ADHD group and minimize group differences in performance. If instead, they reflect limitations during maintenance, delayed recall should selectively impair performance in the ADHD group, but not in the control group. Results showed a benefit of the slow presentation for both groups and an impairment in the delayed condition only for ADHD participants: These results suggest that the working memory deficits in ADHD are related to maintenance mechanisms instead of encoding deficits.

Complementarily to the results of Narimoto et al. (2018), a study by Weigard and Huang-Polock (2017) suggested that slower processing speed is a plausible cause of working memory deficits in ADHD, due to its effects on the temporal dynamics of

attentional refreshing. The latter authors designed a complex span task that manipulated participants' processing speed so that in one condition their processing speed was markedly slower.[13] The slower condition yielded higher cognitive loads and consequent lower memory spans for both ADHD and typically developing participants, which is in line with the predictions of the TBRS model. Weigard and Huang-Pollock (2017) argue that a slower processing speed causes attention to be occupied by the processing task for longer periods, preventing its use to refresh items in the memoranda. On the contrary, a fast processing speed "frees" attention sooner, allowing attentional refreshing to take place. Following the reasoning, children with ADHD have impaired working memory because children of their slower processing speeds.

Both the studies from Narimoto et al. (2018) and Weigard and Huang-Pollock (2017) suggest maintenance as the loci of the attentional deficit in working memory in ADHD. We add two more potential loci to the ones mentioned above. First, children with ADHD might not be able to allocate attention to relevant information and thus face difficulties in selectively maintaining relevant items. Second, they might not be able to switch attention between processing and maintenance demands during working memory tasks, thus being unable to refresh items, as predicted by the TBRS model. The latter view is an extension of the proposition that children with ADHD do not use attention to rehearse memory items during maintenance. In sum, there is a gap in the literature on which working memory mechanism(s) is (are) impaired in ADHD, and research on this clinical population can provide fruitful knowledge about the structure and functioning of working memory.

3.2. Objectives and hypotheses

The goal of this thesis was twofold: 1) to investigate the involvement of attention in working memory, as predicted by the TBRS, by testing a clinical population that is acknowledged for having a pervasive attentional deficit and impairments in working

[13] The authors manipulated the processing speed by varying the difficulty of the processing task and using a diffusion model fit to estimate participants' drift rates (i.e., the average speed of the decision-making process), an index of processing speed. Participants should to make difficult or easy numerosity judgements between each memory item of a complex span task. Trials with difficult numerosity judgements caused slower drift rates (i.e., longer decision-making processes, thus slower processing speeds) than trials with easy numerosity judgements. The authors then grouped trials in two conditions corresponding to fast and slow processing speeds: fast drift rate (easy numerosity judgements) and slow drift rate (difficult numerosity judgements), respectively.

memory functioning; 2) to elucidate what causes working memory deficits in this population.

We will use two different approaches to assess attentional processes in working memory. The first approach taps attentional allocation and uses spatial cues to bias attention towards a target in the perceptual and/or memorized space. This approach evolved from the predictive cueing paradigm proposed by Posner in the 1980s and later adapted to the domain of working memory by Landman et al. (2003) and Nobre and collaborators (Griffin & Nobre, 2003; Lepsien & Nobre, 2006). The cueing paradigm considers attention as a selective focus that prioritizes information at a given spatial location; it is mostly used to study memory for visuospatial content. It is anchored in a large body of evidence showing that spatial attention is essential to bind visual features and create unified mental representations of objects (Nissen, 1985; Treisman, 1988; 1998; Treisman & Gelade, 1980).

The second approach considers attention as a control mechanism and uses the complex span paradigm to assess the ability to switch attention between tasks, namely maintaining and processing items in working memory. This approach is in line with the theoretical scope of the TBRS model and allows us to directly manipulate the cognitive load of a task to test the prediction that a higher cognitive load causes worse performance due to temporal constraints to the use of attentional refreshing.

This thesis comprised one experiment using the cueing paradigm to assess attentional orienting to relevant stimuli and mental representations (Experiment 1) and two experiments using the complex span paradigm to assess the use of attentional refreshing and the effect of the cognitive load on working memory capacity (Experiments 2 and 3). Our main hypotheses are that children and adolescents with ADHD cannot orient attention nor perform attentional refreshing to maintain information in working memory as effectively as their typically developing peers. The specific hypotheses and predictions for each experiment will be explained in the following chapters.

In Chapter 4, we present an overview of the study, from the recruitment of participants to the organization of the experimental sessions. Then, we present Experiments 1, 2, and 3 in Chapters 5, 6, and 7, respectively. Finally, in Chapter 8, we discuss the implications of this thesis to the understanding of ADHD and, ultimately, the functioning of working memory.

Part II

Experimental studies

Chapter 4

General method

4.1. Participants

We recruited 15 children and adolescents diagnosed with ADHD to participate in this study (4 females, mean age = 13.17 years, SD = 1.7) and 67 typically developing children (40 females, mean age = 12.6 years, SD =2.2) aged between 10 and 16 years. After exclusions, the control group included 49 participants (30 females, mean age = 12.8 years, SD = 1.7). The ADHD group included participants diagnosed with the three clinical subtypes (5 participants in the inattentive subtype, 4 in the hyperactive-impulsive subtype, and 6 in th combined subtype). The choice for the age range 10-16 years was based on evidence that attentional orienting in working memory and the use of attentional refreshing are mature from the beginning of adolescence in typically developing individuals (Barrouillet et al., 2009; Shimi et al., 2014; Shimi & Scerif, 2017). Since our main hypothesis is about the difference between ADHD and typically developing children in the use of attentional refreshing for maintaining information in working memory, testing children older than 10 years is adequate.

The inclusion criteria for the ADHD group were: 1) having a formal diagnosis based on the DSM-IV or DSM-5 and done by a general practitioner, a pediatrician, a psychiatric doctor, or a neurologist, and 2) pertaining to the age group. All clinical subtypes (inattentive, hyperactive/impulsive, combined) were included. The exclusion criteria were the presence of supplementary diagnoses of intellectual disability and/or any other neurologic/psychiatric disorder. For participants who were under pharmacological treatment, the inclusion criterion was the intake of a psychostimulant, and the exclusion criterion was the intake of a non-psychostimulant medication. For the control group, the inclusion criteria were age and sex-matched to the clinical group. The exclusion criteria were: 1) the presence of a formal diagnosis of ADHD, 2) the presence of ADHD symptoms and 3) the presence of an intellectual disability and/or other neurologic/psychiatric disorder.

Table 2 shows the characterization of the ADHD group and Annex 1 shows the complete characterization of our sample. The clinical subtypes shown in Table 2 reflect the symptoms manifest at the time of the survey (up to six months before the completion of the questionnaires), thus not necessarily the clinical subtype specified by the medical doctor when the participant was first diagnosed and informed by parents during recruiting. As extensively discussed in Chapter 2, it is common that the predominant symptomatic dimension of ADHD changes during adolescence, with hyperactivity/impulsivity losing ground to inattention. For this reason, we opted to present in Table 2 the clinical subtypes of participants according to the results of the symptoms inventories used during recruiting.

Table 2

Characterization of participants in the ADHD group

Sex	Age	ADHD Subtype	*T*-score Inattention	*T*-score Hyperactivity-impulsivity	Overal probability
F	11.2	Combined	90	90	0.99
F	15.4	Hyperactive	73	90	0.97
F	12.0	Inattentive	90	45	0.71
F	15.9	Inattentive	90	47	0.87
M	12.8	Combined	80	72	0.99
M	13.3	Combined	85	89	0.99
M	13.5	Hyperactive	57	67	0.41
M	10.7	Inattentive	83	63	0.87
M	15.7	Combined	90	81	0.99
M	11.5	Combined	75	90	0.99
M	13.9	Combined	75	90	0.99
M	13.1	Inattentive	66	40	0.51
M	14.6	Inattentive	44	45	0.11
M	11.1	Hyperactive	53	67	0.56
M	12.7	Hyperactive	65	90	0.91

Note. The columns *T*-score inattention, *T*-score hyperactivity-impuslvitiy, and Overal probability contain the scores in the Conners-3 Parent subtests. The ages are represented in years.

Our study protocol was registered and authorized by the local ethics committee, the CER-VD (Commission cantonale d'éthique de la recherche sur l'être humain – Vaud) under the number 2019-02325. All parents/legal guardians gave written consent to their

child's participation in the study, and participants above 14 years old gave their written consent as well, according to Swiss legal requirements (see Annex 2 for the consent forms).

4.2. Materials

Our study comprised a first stage of recruiting and selecting participants and a second stage of participating in computerized experiments. We will first present the instruments we used to select participants during recruitment and then we will present the apparatus used for the experimental tasks.

4.2.1. Questionnaires and scales

The medical history and the presence of psychiatric diagnoses and symptoms in both groups were assessed prior to participation in the experiments. For this purpose, parents and/or legal guardians filled in a set of questionnaire and scales described below.

Medical History Questionnaire. This questionnaire was specifically developed by us for the purposes of our research. It contained five questions about the presence of formal psychiatric diagnoses and seven questions about the intake of medication (Annex 3).

Conners-3 Parent. This scale assesses ADHD symptoms in individuals from 6 to 18 years old. The Conners-3 Parent is part of the Conners Scale, 3rd edition (Conners, 2008) and it is specifically addressed to parents. Each item of the Conners-3 Parent contains a description of a behaviour or characteristic (e.g., "Has bad orthography", "Moves constantly", "Do not pay attention to details, makes inattention mistakes) whose frequency must be rated on a five-point Likert scale from 'never' to 'very often'. The *T*-scores and percentiles are calculated for internalizing symptoms, hyperactivity/impulsivity, learning problems, executive functioning, defiance/aggression, peer relations, and the incidence of symptoms of ADHD according to the DSM-IV-TR (American Psychiatric Association, 2000).

The Child Behavior Checklist – CBCL. The CBCL (Achenbach and Rescorla, 2001) is another questionnaire addressed to parents. It assesses children's competencies, emotional and behavioural problems associated with ADHD. It has a list of 112 characteristics and behaviours of children and adolescents, to be judged by parents in a three-point Likert scale from "Does not apply" to "Always or most of the time true". The

answers are grouped in two major syndromes scales: "internalizing problems" (combines answers related to anxious, depressed, and withdrawn behaviours and somatic complaints) and "externalizing problems" (combines rule-breaking and aggressive behaviours). The scores in each scale and subscale are then compared to normative through the calculation of *T*-scores and percentiles.

The ADHD Rating Scale. (ADHD-RT, DuPaul, Power, Anastopoulos, & Reid 1998) is a scale measuring the frequency of ADHD symptoms at home in the past six months based on DSM-IV (APA, 2000) criteria. It is composed of two subscales, one about inattentive symptoms and other about hyperactivity symptoms. The frequency of each symptom is rated by parents in a four-point Likert scale from "rarely or never" to "very often" and then the answers are independently graded by a clinician and/or researcher.

The Behavior Rating Inventory of Executive Function – BRIEF. The BRIEF (Gioia, Isquith, Guy, & Kenoworthy, 2000) is a questionnaire measuring executive functioning in everyday life. The BRIEF includes 85 questions on eight sub-domains, clustered in two groups of scales: behavioural regulation (includes "inhibit", "shift", "emotional control"), and metacognition (includes "initiate", "working memory", "plan/organize", "organization of materials" and "monitor"). The raw scores are transformed into *T*-scores, percentiles and 90% confidence intervals according to the age group and gender of the child/adolescent.

4.2.2. Apparatus

The experiments were programmed and implemented with the software PsychoPy v.2020.1.3 (Peirce et al., 2019) on a laptop computer (HP Probook 440 G6). The screen measured 14 inches, the resolution was 1920 x 1080 pixels, and the refreshing rate was 60 Hz. Response collection was implemented via mouse using a wireless (Bluetooth) device in Experiment 1.

4.3. General procedure

The research was conducted in two stages: an initial stage of recruitment, contact with families and selection of participants, and a second stage in which participants took part in the experiments. We will describe them separately.

4.3.1. Recruitment and selection of participants

We advertised our study on social media and through flyers and posters (Annex 4) on health clinics, psychological services, pharmacies, schools, and a parents' association in the French-speaking cantons of Switzerland (ASPEDAH, L'Association Suisse romande de Parents d´Enfants avec Déficit d´Attention, avec ou sans Hyperactivité). The participants' parents or legal guardians then contacted us to state their interest in taking part as volunteers in our study. After a preliminary meeting with the researcher to the research project in detail, the parents/legal guardians received a set of documents by post. This set of documents contained the consent form, the set of questionnaires and scales, and a stamped, self-addressed envelope so they could send back their written consent and the completed questionnaires. For participants aged between 14 and 16 years, we asked for both theirs and their parents/legal guardian's written consent.

For the families in the ADHD group, the set of questionnaires and scales was composed of the Child Behavior Checklist (CBCL, Achenbach & Rescorla, 2001); the Conners-3 Parent version (Conners, 2008), the ADHD rating scale (ADHD-RS, DuPaul, Power, Anastopoulos, & Reid 1998, French version by Mercier, Roche, Gaillard, et al. 2016), the Behavioral Rating Inventory for Executive Function (BRIEF, Gioia, et al., 2000), and the questionnaire about the medical history of the child. For the families in the control group, the set of questionnaires was composed of the CBCL (Achenbach & Rescorla, 2001), the Conners-3 parent version and the questionnaire on the medical history.

The parents/legal guardians returned the completed consent forms and questionnaires by post. After correcting the scales and questionnaires, we contacted the parents/legal guardians to inform them if their child had fulfilled the inclusion criteria and to invite them to participate in the experiments. In case of a fortuitous discovery during this screening phase, families were referred either to their own treating doctor or to our referring doctor, according to their previous choice and consent. There were three possibilities of fortuitous discoveries during the screening phase: a) a participant who had previously been diagnosed with ADHD does not reach criteria for symptoms in the scales and questionnaires; b) a participant without any psychiatric diagnosis is suspected of having ADHD symptoms or other psychiatric disorders; c) a participant with a previous ADHD diagnosis but no comorbidity is suspected of having another comorbid disorder.

The medical consultations after referral were out of the scope of the study and were a supplementary resource for families whose children needed further medical investigation. The families were, therefore, free to schedule a consultation with our referring doctor if they wished so.

4.3.2. Experimental sessions

Each participant took part in three individual experimental sessions, each lasting about 30 to 40 minutes. Participants in the ADHD group were asked to cease the intake of psychostimulant at least 24 hours before each experimental session. In cases where participants took medication only on school days, the sessions were scheduled on weekends and/or school holidays to minimize the impact on the family's routine. The experimental sessions were carried out in a silent room in a place chosen according to the family's convenience (e.g., the family's house, a public library, the University of Fribourg)[14]. Participants in the control group were tested individually in a silent room at school during school hours. The computer was placed in front of the participant from about 60 centimetres. Participants in the ADHD group could take short breaks during the experimental sessions.

Experiments 1, 2, and 3 and their specific procedures will be presented in separate chapters of this thesis, followed by the respective results and discussions. Experiment 1 consisted in a cued colour recognition task used to test attentional orientation during the processing stages of encoding and maintenance. Experiments 2 and 3 consisted of complex span tasks used to test specific predictions on the effect of the cognitive load and the use of attentional refreshing in working memory. If children and adolescents present difficulties to orient attention to the contents of working memory and to employ it to maintain the memoranda, then we expect interactions between the factor group and the experimental conditions in Experiments 1, 2, and 3. Our predictions and hypotheses for each experiment will be presented next.

[14] Since this doctoral research was conducted during the Covid-19 pandemic, the hygiene procedures advocated by the Swiss public health authorities were strictly followed during the experimental sessions. All participants were asked to disinfect their hands with hydroalcoholic solution at the beginning and at the end of the experimental session, and the participants older than 11 years were asked to wear a mask. The experimenter herself wore a mask during every contact with the participants and their families. The experimenter remained with the participant during the whole experimental session, at a distance of at least 1.5 m after she gave the instructions. Whenever possible, the room's windows remained opened during the sessions. A disinfection routine of the touchable surfaces (table, computer mouse, and keyboard) was carried out by the experimenter at the beginning and at the end of each experimental session.

Chapter 5

Experiment 1: The cued colour recognition task

Experiment 1 examined the visuospatial domain of WM, and it was designed to tackle attentional allocation during the processing stages of encoding and maintenance. The experiment comprised a colour recognition task combined with the predictive and the retroactive cueing paradigms (Posner 1980, Griffin & Nobre, 2003; Landman et al., 2003). The predictive cueing paradigm consists of presenting a cue (location cues, in the classical experiments first reported by Posner, 1980) that anticipates the target, prior to stimuli presentation, thus biasing the subject's perceptive attention to the relevant item in the ongoing task. The retroactive cueing paradigm, in turn, consists of presenting a cue after the offset of the stimuli, thus biasing attention retrospectively to a specific item in the memoranda (the cued item). Predictive cues (henceforth pre-cues) bias encoding before the presentation of the items, limiting the number of items encoded into WM, whereas retroactive cues (henceforth retro-cues) signals the item to retrieve after the presentation of the stimuli.

Typically, experiments using cueing paradigms employ location cues, but there are solid results in the literature on the efficacy of other cue types, like feature-based cues (colour, shape) and semantic categories (Heuer & Schubö, 2016; Loaiza & Camos, 2018; Niklaus et al., 2017; Ye et al. 2016). In Experiment 1, we opted to use location cues because our experimental design was based on a study by Shimi et al. (2014), who also used location pre- and retro-cues. Moreover, there is a large body of knowledge dating back to the 1990's that show that individuals with ADHD have no difficulties in orienting perceptual attention to location cues (see Huang-Pollock & Nigg, 2003, for a meta-analytical review), which controls for potential confounding effects of the cue type.

In the typically developing population, retro-cues enhance working memory performance to a similar extent to predictive cues recognition and change detection tasks (Griffin & Nobre, 2003; Lepsien & Nobre, 2006), a phenomenon known in the literature

as the "retro-cue effect". The retro-cue effect has been reported in healthy young adults and children from age 7 (Astle et al., 2012; Guillory et al., 2018; Shimi, Nobre et al., 2014; Shimi & Scerif, 2017), healthy elderly people (Gurau et al., 2020; Souza, 2016; Strunk et al., 2019), and patients with mild cognitive impairment (Newsome et al., 2015). The retro-cue effect is acknowledged to reflect the human ability to deploy attention to mental representations stored in working memory, boosting the maintenance of the cued item and thus improving its retrieval in comparison to non-cued items. To the best of our knowledge, no studies investigated if the retro-cue effect replicates in children diagnosed with ADHD.

Our Experiment 1 aims to investigate if children and adolescents with ADHD will benefit from spatial retro-cues during a colour recognition task. Because they present a chronic deficit in attentional control, we hypothesized that they will not be able to deploy attention retrospectively to boost working memory performance and therefore will not be prone to the retro-cue effect. According to this hypothesis, we expected the following results in Experiment 1, from the most general to the most specific: 1) performance in the ADHD group would be worse than in the control group, in all experimental conditions; 2) the control group will benefit both from predictive cues and retrospective cues; 3) the ADHD group will only benefit from predictive cues, but not from retroactive cues.

5.1. Method

5.1.1. Participants

Fifteen participants took part in Experiment 1 in the ADHD group (4 females, mean age = 13.1 years, SD = 1.7) and nineteen participants took part in the control group (13 females, mean age = 13.0 years, SD = 1.6). After the discarding of the data from three participants because they were outliers in the first quartile of the data, the control group included 16 participants (11 females, mean age = 13.0 years, SD = 1.5).

The mean *T*-scores in the Conners-3 subscale of inattention were 71.06 (SD = 19.3) in the ADHD group and 55.1 (SD = 9.6) in the control group. The *T*-scores in the Conners-3 subscale of hyperactivity-impulsivity were 71.06 (SD = 19.93) in the ADHD group and 55.18 (SD = 9.6) in the control group. The clinical threshold for the Conners-3 subscales is a *T*-score equal to or above 61, corresponding to the 85th percentile. We tested whether the *T*-scores of inattention and hyperactivity/impulsivity differed between the groups of participants in a preliminary analysis. We used Bayesian independent samples *T*-tests and

set the alternative hypotheses as *T*-scores$_{(ADHD)}$ > *T*-Scores$_{(controls)}$. This analysis showed very strong evidence of a group difference regarding the *T*-scores of inattention (BF_{10} = 6959.6) and hyperactivity (BF_{10} = 13.82), confirming that clinical symptoms were present in participants diagnosed with ADHD and absent in controls[15]. The complete characterization of the sample in Experiment 1 can be found in Annex 1.

5.1.2. Material and stimuli

The stimuli pool consisted in 64 coloured animals, resulting from the combination of eight flat silhouettes of animals (rabbit, dog, duck, pigeon, fox, penguin, cat, and poodle dog) and eight colours. The colours and their RGB coordinates were the following: green (0, 128, 0), blue (0, 0, 255), orange (255, 165, 0), yellow (255, 255, 0), red (255, 0, 0), white (255, 255, 255), pink (255, 105, 180), and violet (148, 0, 211). The flat silhouettes were inscribed in a square with sides measuring 6 cm and had a transparent background.

The central fixation point was a sum symbol in the font "Calibri", letter height set to 0.1 in PsychoPy's settings. For the predictive and retroactive cues, we used central white arrows pointing to four possible locations: top right, top left, bottom right, and bottom left in relation to the centre of the screen. The cues (both predictive and retroactive) were inscribed in a square with sides measuring 4 cm. For the control condition, we used a central white square with sizes measuring 4 cm. The central white square did not provide any spatial information and will henceforth be called "neutral cue". The background colour of the window was grey (RGB: 175, 175, 175).

5.1.3. Procedure

Each trial began with the display of the central fixation cross during 1000 ms. In the control condition, the fixation cross was followed by the presentation of the neutral cue during 300 ms. After the offset of the neutral cue, the fixation cross reappeared in the centre of the screen for 1000 ms. This was followed by the display of four stimuli (the same animal

[15] During the analysis, we noticed that parent's responses in the Conners-3 questionnaire were incongruent for some participants in the control group, e.g., a child with very high scores of hyperactivity-impulsivity but scores for aggressivity and peer relations in the normal range (or vice versa), followed by very modest estimations of the damage caused by her behaviour in daily life (e.g., a child that is rated as inattentive above percentile 95 percentile but whose inattention never affects the family and academic life negatively). This not rarely yielded results above the clinical threshold advocated by the instrument. For this reason, we compared those limit cases in the control group to the cases in the normal range for all subscales. We did not find any differences between them, so we decided to keep those participants in the control group. This preliminary analysis is in Annex 5

silhouette but in different colours) around the fixation cross for 350 ms. Each stimulus was placed at one vertex of an imaginary square centred at the fixation cross and with sizes measuring 11.6 cm. After the offset of the stimuli, the fixation cross was displayed for 1000 ms and then followed by the presentation of a second neutral cue during 300 ms. After the offset of the neutral cue, the fixation cross was displayed for another 1000 ms and followed by the exhibition of the probe at the centre of the screen. The probe could be either the silhouette of the animal in one of the four colours present in the trial or in a different colour, not seen in the trial. The participant should recognize the colour of the probe by responding via mouse press (right button for "Yes, known colour", and left for "No, unknown colour"). We put coloured stickers (green for "yes" and red for "no") on the mouse buttons to make response easier for children. The probe remained visible until a response was given. An intertrial screen with the sentence "*Appuie sur espace pour continuer*" (Press space to continue) was displayed at the end of each trial, allowing participants to control the pace of the experiment.

In the predictive cue condition, the first neutral cue was replaced by a central arrow pointing to one of the four locations around the fixation cross. The second neutral cue was exhibited at the same time point as in the neutral condition. In the retro-cue condition, the first neutral cue was displayed at the same time point as in the neutral condition, but the second was replaced by a central arrow pointing towards one of the four possible locations. We used the white squares as neutral cues at the same time point as the predictive cues and retro-cues in the control condition to control for non-spatial alerting effects of the cues. This choice was made based on the experiments reported by Shimi et al. (2014). Figure 4 shows the order of the events in a trial in each experimental condition.

The cueing conditions (predictive cue, retro-active cue, neutral cue) were manipulated between blocks of trials and the order of presentation of the blocks was counterbalanced between participants. Each block had 48 trials and was preceded by 8 practice trials. The probe colour was present in half of the trials (positive trials) and absent in the other half (negative trials) in a block. In the predictive and retroactive cueing conditions, the cues were 100% valid in positive trials, i.e. they always pointed to the location occupied by the probed colour. In negative trials, the probe was always a colour absent in the trial.

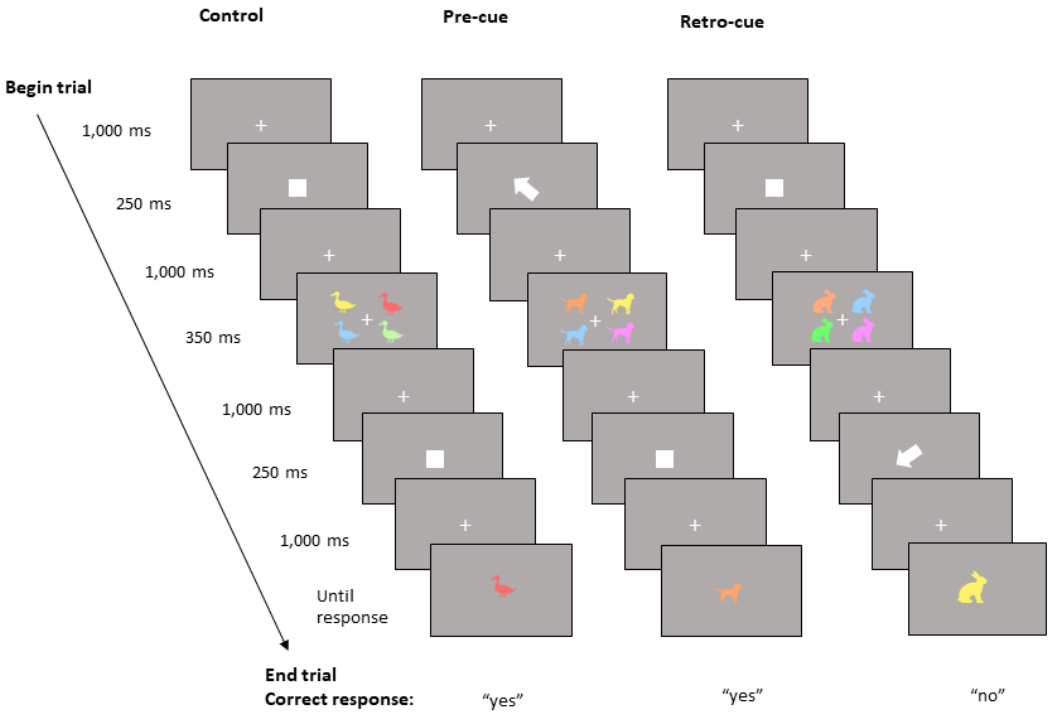

Figure 4. The cued colour recognition task. The cue was 100% valid in all experimental conditions. The colour probe was present in the array in 50% of the trials. In the trials exemplified in the control and pre-cue conditions, the expected correct response is yes (hit). In the trial exemplified in the retro-cue condition, the expected correct response is no (correct rejection).

At the beginning of each block, the experimenter explained the procedure to the participant while showing a scheme of the order of the events in a trial (Figure 4). The child participant then performed the practice trials. Written feedback - the words "*C'est juste!*" (Correct!) and "*C'est faux*" (Incorrect) appearing at the centre of the screen - was given at each trial during the practice trials. At the end of the practice trials, the question "*Tu te sens prêt pour commencer?*" (Do you feel ready to begin?) appeared on the screen and the participant could choose to proceed to the block of trials or to repeat the practice trials.

5.1.4. Data analysis

For each condition and each participant, we calculated the discrimination indexes (*d'*) by applying the formula $z(H) - z(F)$, where z corresponds to the Z-transformed scores of the hit rate (H) and false alarm rate (F) (Macmillan & Creelman, 2005). We applied the correction formula proposed by Snodgrass and Corwin (1988) to rule out extreme values of one and zero that render the Z-transformation impossible.

We compared the means between groups and experimental conditions by using a Bayesian repeated-measures ANOVA with the group (ADHD vs. controls) as a between-subjects factor and the cueing condition (neutral, pre-cue, and retro-cue conditions) as a within-subjects factor. For this analysis, we set the prior odds for each model as equivalent, that is, $P_{(M)} = 0.2$ for all models. This choice was done based on the absence of results reported with Bayesian statistics for this type of experimental design and population in the literature.

5.2. Results

In the ADHD group, the mean of the *d'* was 1.91 (SD = 0.79) in trials with neutral cues, 3.17 (SD = 0.83) in trials with pre-cues, and 2.82 (SD = 0.77) in trials with retro-cues. In the control group, the mean of the *d'* was 2.43 (SD = 0.68) in trials with neutral cues, 3.45 (SD = 0.44) in trials with pre-cues, and 3.03 (SD = 0.81) in trials with retro-cues. Figure 5 shows these results. On average, the cueing benefit provided by pre-cues was 1.26 (SD = 0.92) in the ADHD group and 1.01 (SD = 0.69) in the control group. As for retro-cues, their cueing benefit were 0.91 (SD = 0.77) in the ADHD group and 0.6 (SD = 0.91) in the control group.

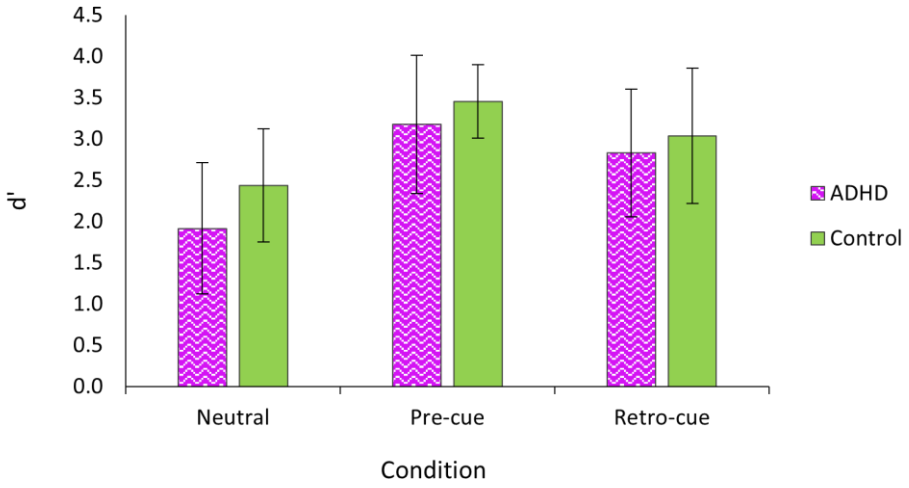

Figure 5. Means of the *d'* per group and experimental conditions in Experiment 1. The vertical bars represent the standard deviations.

Although the controls outperformed the ADHD group in all experimental conditions, the Bayesian ANOVA showed that the best model accounting for the data was the model including only the main effect of the cueing condition, $BF_{10} = 4.624 \times 10^7$ (error = 0.72, indicating very strong evidence of this model against the null model. According to this model, participants both in the ADHD and control groups equally benefited from pre-cues and retro-cues. The second-best model was the model including the main effects of the cueing condition and the group, $BF_{10} = 4.235 \times 10^7$, error = 2.01. According to this model, both ADHD and controls benefited from predictive and retro-cues, with controls outperforming the ADHD group in all conditions.

Our hypothesis for Experiment 1 predicted an interaction between the factors group and cueing conditions, with the ADHD group benefiting from pre-cues but not from retro-cues, whereas controls would benefit from both. The Bayes factor for the full model was far behind the two best models presented above in terms of the predictive power of our data, with a BF_{10} of 1.07×10^7 (error = 2.7). Compared to the other two models, we do not have strong enough evidence to support the interaction between cueing conditions and group. Concerning the main effects of the cueing conditions and the group, we decided to further examine the BF_{incl} and BF_{excl} of each factor as the Bayes factors of the two best models did not differ strongly.

Again, we found very strong evidence in favour of the inclusion of the cueing effects in the model ($BF_{incl} = 3.843 \times 10^7$). As for the main effect of the group, the evidence for its exclusion was not decisive, with a BF_{excl} of 1.32. In regard to the predicted interaction between cueing conditions and group, the evidence in favour of its exclusion in the model was higher than for the exclusion of the group solely, but it was nevertheless not decisive as well, with a $BF_{excl} = 2.07$. Taking all the Bayes factors presented so far, our interpretation of the results was that the cueing condition accounted for nearly all the difference between the means in the *d'*, with a trend of a group effect causing ADHD to perform slightly worse than controls.

In order to examine this trend, we ran sequential Bayesian analyses comparing the two groups across conditions with independent samples *T*-tests. This type of analysis allowed us to examine how the posterior odds and the Bayes factors change at every new

observation in the data. We set the alternative hypothesis as $d'_{(ADHD)} \neq d'_{(Control)}$. Figure 6 shows the results of these analyses for each experimental condition.

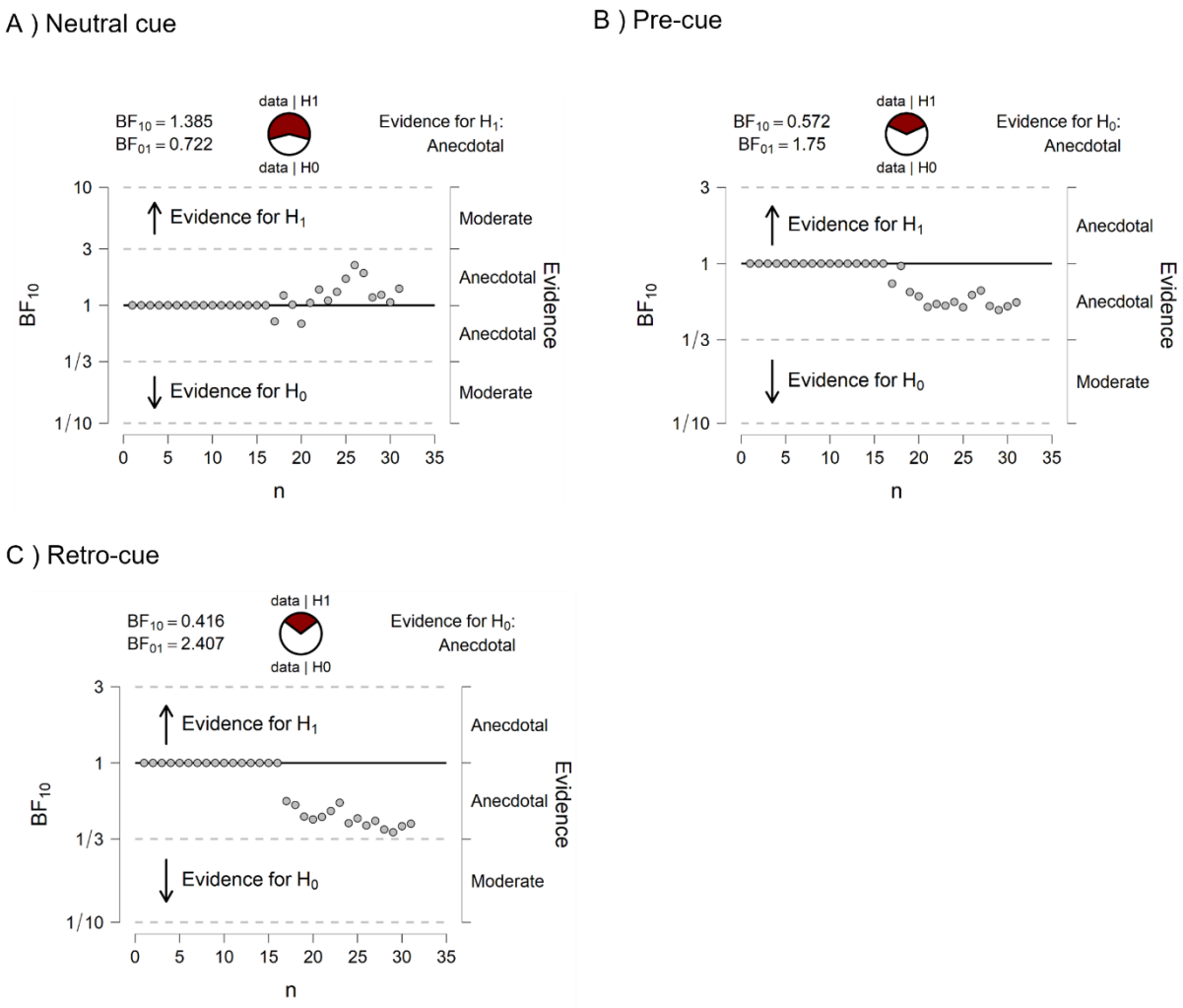

Figure 6. Sequential Bayesian analysis of the *T*-tests comparing the *d'* of the groups in the neutral cue (A), pre-cue (B), and retro-cue (C) conditions. In the three panels, the group of points to the right side of the graph represent the cumulative changes in the Bayes factor of the alternative hypothesis, at every addition of a data point corresponding to the *d'* of an ADHD participant.

Except for the neutral cue condition, in which the cumulative evidence remains in-between the null hypothesis (no group difference) and the alternative hypothesis, there was no big fluctuation with the addition of participants in the sequential Bayes factor. In the pre-cue condition, the sequential Bayes factors did not reach the threshold between anecdotal and moderate evidence in favour of the null. In the retro-cue condition, there

was a trend towards moderate evidence in favour of the null. Either way, the cumulative BF_{10} consistently remained below 1 in the pre-cue and retro-cue conditions, which constitutes evidence of an absence of a group effect.

To better visualize the absence of this group effect, we plotted the *d'* of each participant in the ADHD group against the 95% confidence intervals of mean *d'* in the control group (Figure 7). The plots allowed us to visualize how participants in the ADHD group fall within the mean performance of the control group. Again, the distribution of the data points showed that there was an overlap between the *d'* of the ADHD participants and the confidence intervals of the control group in all experimental conditions, supporting the absence of the group effect and the hypothesized interaction. As the figures above make clear, performance in the ADHD group greatly varied across all experimental conditions. Given that our choice for a broad age group in our inclusion criteria (ages between 10 and 16 years), there was great variation in the *d'* of both groups caused by the heterogeneity in participants' ages. Specifically, about the variability in performance in the ADHD group, we supposed that the inclusion of participants from different clinical subtypes in the ADHD caused great heterogeneity in the clinical levels of inattention of participants, as measured by the symptom questionnaires. On the one hand, ADHD participants with higher levels of inattention could present smaller cueing benefits compared to those with lower levels of inattention. On the other hand, it could also be that their baseline performance (i.e., performance in neutral cue trials) would be at floor level and therefore any increase caused by the cueing conditions would yield larger cue benefits compared to those of participants with fewer symptoms of inattention.

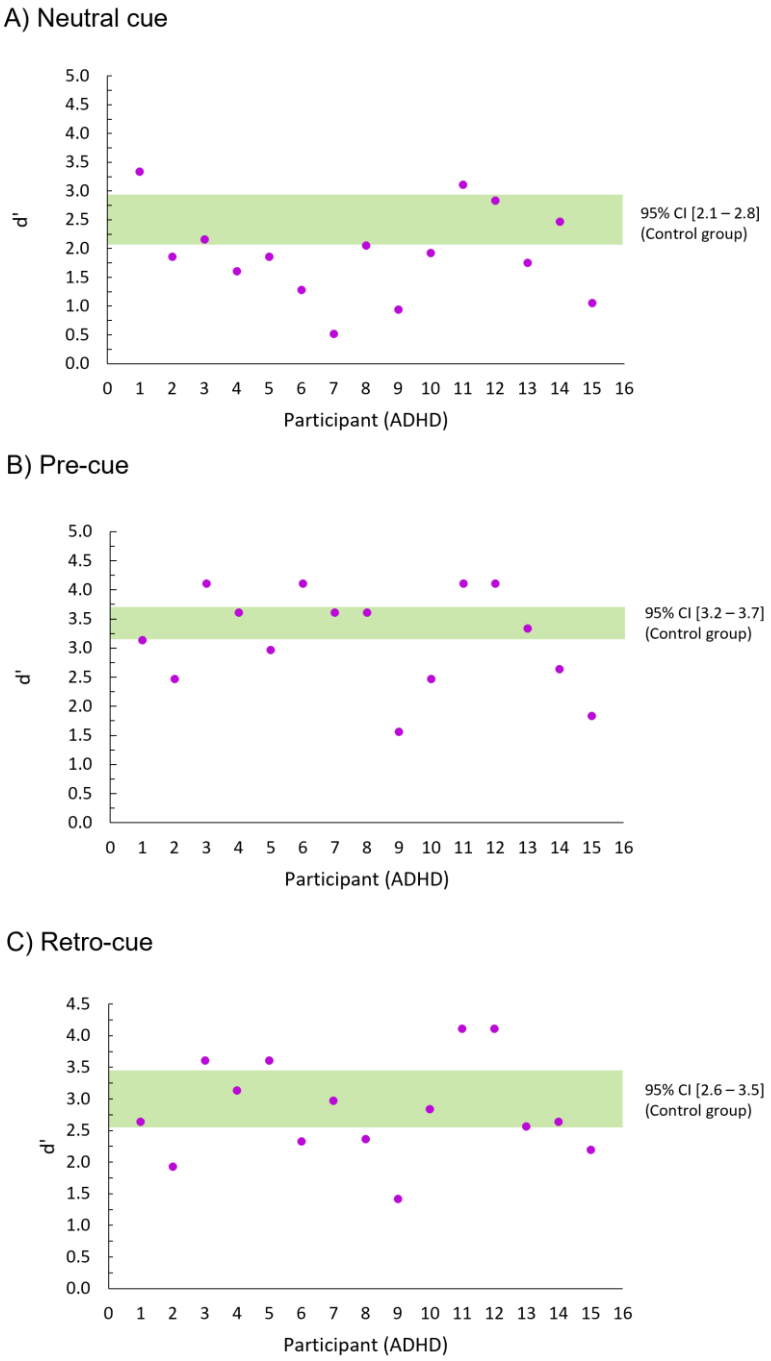

Figure 7. Individual *d'* in the ADHD group in the neutral cue (A), pre-cue (B), and retro-cue (C) conditions. The green zones represent the 95% confidence intervals of the control group.

To better account for the diversity of the clinical profiles of our participants, we included the *T*-scores of inattention and hyperactivity in the Conners-3 subscales as covariates in a Bayesian ANOVA. This analysis included both ADHD and control participants. The best model was the one including the main factors cueing condition and inattention (BF_{10} = 1.092 x 10^9), and the second-best included the effect of cueing

condition, inattention, and hyperactivity (BF_{10} = 4.806 x 10^7). Nevertheless, the examination of the BF associated with each factor revealed decisive evidence in favour of the addition only of the factor cueing condition (BF_{incl} = 6.285 x 10^7), but not inattention (BF_{excl} = 0.56) nor hyperactivity (BF_{excl} = 1.68).

Finally, we ran Bayesian Pearson correlations between the *T*-scores of inattention of all participants and the cueing benefits in the pre-cue and retro-cue conditions. Table 3 shows the correlation between the variable pairs. The negative correlation between inattention and the retro-cue benefit was negligible (r = 0.027), reinforcing our interpretation that the *T*-score of inattention does not reflect one's ability to make use of retro-cues. The positive correlation between inattention and the pre-cue benefit was also negligible (r = 0.15). To end, we found very strong evidence (BF_{10} = 50.615) of a moderate positive correlation (r = 0.57) between the cueing benefits of pre-cues and retro-cues. Taken together, these results indicated that regardless of the *T*-scores of inattentions in the Conners-3 subscales, the cueing benefits provided by pre-cues and retro-cues were moderately correlated in children in our sample, both ADHD and controls.

Table 3

Bayes factors of the Pearson correlations between inattention and cueing benefits in Experiment 1

		T-score inattention	Pre-cue benefit	Retro-cue benefit
T-score inattention	Pearson's *r*	—		
	BF_{10}	—		
Pre-cue benefit	Pearson's *r*	0.153	—	
	BF_{10}	0.309	—	
Retro-cue benefit	Pearson's *r*	-0.027	0.573	—
	BF_{10}	0.226	50.615	—

5.3. Discussion

The results of Experiment 1 showed that both pre- and retro-cues benefited participants in both groups of participants and that controls outperformed ADHDs in all experimental conditions. Albeit recognition memory in the control group was higher than

in the ADHD group, the Bayesian ANOVA comparing the means of the *d'* across conditions and groups showed that there was no evidence of a group difference in cueing benefits in Experiment 1. All the analyses presented above supported a strong effect of the cueing manipulation and an absence of a group effect and their hypothesized interaction. Although there was a small difference between the two best models, the results of the sequential Bayesian analysis supported our choice for the model predicting only a main effect of the cueing condition to explain the data.

The absence of a group effect in Experiment 1 was an unexpected result for us, given all the literature reporting working memory deficits in ADHD discussed in Chapter 2. We partially account this result to our heterogeneous sample regarding the age and symptomatic levels of participants. Ideally, future research should include a clinical group containing only participants diagnosed with the inattentive and combined subtypes of ADHD, and the age criteria should be narrowed to 10-to-11-year-olds, as in the study by Shimi et al. (2014). This was the age group that has more benefited from retro-cues in their study, with cueing benefits similar to young adults (Shimi et al., 2014).

Through pure observation, we noticed that children in the ADHD group behaved differently in the experimental setting according to their age and clinical subtype. Although presenting higher levels of inattention (thus purportedly facing more difficulties in the task), children in the inattentive subtype were more resistant to the effects of fatigue and endured longer in the task. In their turn, hyperactive-impulsive participants are less resistant to frustration and sometimes abandoned the task by simply refusing to engage, leaving the experimental setting, or threatening to turn off the computer/press the "ESC" key during the task. To cope with these situations, the experimenter negotiated small breaks with the participants and sometimes parental intervention was needed to bring hyperactive-impulsive children back to the testing room. Also, older participants tended to be more confrontational and sometimes responded recklessly to the task, requiring interventions from the experimenter. We believe that more control over the diagnostic subtype and the age of participants is necessary to completely rule out the existence of a group effect between ADHD and controls in the cued recognition task.

The most important result is the strong effect of the cueing manipulation that enabled pre- and retro-cue benefits in the ADHD group. Regarding pre-cue benefits, Experiment 1 confirmed what has been previously reported in the literature. The presence of cueing benefits in perceptual tasks (i.e., simple target detection) in the ADHD

population has been reported since the 1990s (Huang-Pollock & Nigg, 2003; Jonkman, 2005), therefore it is not a novelty that children with ADHD are subject to cue-induced anticipatory effects of attentional orienting, a phenomenon indexed by the pre-cue condition in our Experiment 1. Moreover, there is meta-analytical evidence (Huang-Pollock & Nigg, 2003) that ADHD is not characterized by a dysfunction in visuospatial orienting[16].

A study by Mazaheri et al. (2010) showed that children with ADHD (ages between 8 and 12 years) benefit from symbolic cues in a cross-modality reaction task. In this study, symbolic valid cues signalled the modality (visual vs. auditory) of the upcoming target in 75% of the trials, and participants were required to answer if a target was a visual or an auditory stimulus. The authors found significant effects of the cue validity upon response accuracy[17] in both groups and sensorial modalities and, as in our Experiment 1, they found no interaction between the group and the cueing effects. Furthermore, the pattern of results for visual targets was similar to our Experiment 1: Despite controls overperforming children in the ADHD group, the difference between the groups did not reach statistical significance in the study of Mazaheri et al. (2010). The pre-cue benefits observed in our Experiment 1 add evidence to the body of research attesting that perceptual attentional orienting is functional in ADHD and shows that, at least in the visuospatial domain, it generalizes to working memory. We are not aware of previous studies that tested pre-cueing effects in the auditory working memory of children with ADHD – a theme that is extraneous to the goals of this thesis. We believe it is an interesting topic for future research.

The interplay between attention and working memory is one of the most important topics in cognitive psychology and the functional similarities between cueing effects in visual working memory and perception have been extensively reported since the seminal works of Nobre and colleagues and Landman et al. (Griffin & Nobre, 2003; Landman et al., 2003; Lepsien & Nobre, 2006) on the retro-cue effect. In the past two decades, the retro-cue effect has been reported in typically developing populations of adults, children, and elderly, and in clinical populations of people with hearing loss and mild cognitive impairment (Garami et al., Loaiza & Souza, 2018; 2019; Newsome et al., 2015; Shimi, Kuo et al., 2014; Shimi, Nobre et al., 2014; Shimi & Scerif, 2021; Souza, 2016; Strunk et

[16] Huang-Pollock and Nigg (2003) make a caveat that none of studies included in their meta-analysis had large enough samples to control for individual differences among the ADHD group and only one study looked in the inattentive subtype of ADHD. As presented, this thesis partakes of this limitation.
[17] The dependent variable was the percentage of targets correctly detected instead of the *d'*.

al., 2019). Together, these studies seem to point to the generability of the retro-cue effect in working memory, a sign that humans voluntarily use attentional orienting to boost working memory performance from childhood to old age. Whether or not this ability is preserved in populations with a chronic attentional deficit is therefore an important question to understand the mechanics of attentional orienting in working memory.

The retro-cue benefits observed in Experiment 1 suggest that ADHD children are capable retrospectively orient attention in working memory and that this ability is not impaired compared to their typically developing peers. To the best of our knowledge, this was the first time in the literature that the retro-cue effect has been reported in a population of children and adolescents with ADHD. The novelty of our Experiment 1 is, therefore, the observation of a robust retro-cue effect pointing towards a functional process of attentional orienting to the memorized content by a clinical population characterized both attentional-deficits and impairments in working memory.

Chapter 6

Experiment 2: The reading digit span task

Experiment 2 was designed to tackle the use of attentional refreshing in a verbal WM task. It consisted of a complex-span task in which participants were required to memorize increasing sequences of letters and to read aloud digits between the presentation of each letter. The experimental design was based on a previous task reported by Barrouillet et. al (2009). Following their Experiment 1, we manipulated the speed of the digits' presentation in a fixed interval between each letter in the sequence, so that participants performed the task in two different conditions: "fast pace" and "slow pace". The original task in Barrouillet et al. (2009) had four different pace conditions, but we chose to use only two for the sake of simplicity and to avoid a lengthy experimental procedure that could be detrimental to the performance of the ADHD group.

The core element of this experimental manipulation is the prediction of the TBRS model that when performing a dual-task (in this case, remembering the letter series and reading the digits aloud), the participant must divide her attentional resources between maintaining information in WM and processing information for the concurrent task, switching attention between these two tasks. When attention is focused on processing the distracting items, the information held in WM is prone to temporal decay; conversely, memory traces are reactivated when attention is focused on memory traces for their maintenance. According to the TBRS model, one's WM performance depends on the efficiency of this rapid switching of attention between maintenance and processing. The more time one has to employ attention in the refreshing of items in WM, the better will be her recall of information. That said, the purpose of manipulating the pace of the digits' presentation was to introduce two different conditions regarding the possibility of performing attentional refreshing: one in which participants have more free time to refresh the letters, (slow pace condition) and another in which refreshing is impaired by reducing the available time for maintenance activities (fast pace condition).

The main hypothesis of this thesis is that a misuse of attentional resources is an essential part of the WM deficits in ADHD. One potential source of misuse is not being able to use attention to refresh the contents of WM, or, in other words, not being able to perform attentional refreshing. Experiment 2, therefore, was designed to test the prediction that children and adolescents with ADHD cannot use attentional refreshing to maintain WM traces. According to this hypothesis, because only typically developing participants are expected to use attentional refreshing spontaneously, we anticipate that the fast pace condition will be detrimental to memory performance in the control group, but not in the ADHD group. Our predictions are the following, from the most general to the most specific: 1) the mean span of the ADHD group will be lower than the mean span of the control group, due to a general deficit on attention; 2) the deleterious effect of the fast pace condition upon performance in the ADHD group, if any, will be less steep than in the control group.

6.1. Method

6.1.1. Participants

Fifteen participants took part in Experiment 2 in the ADHD group (4 females, mean age = 13.2 years, SD = 1.7 year) and 19 in the control group (13 females, mean age = 13.1 years, SD = 1.8 year). One participant in the control group was an outlier in all the dependent variables, so his data were excluded from the analyses. The final sample included 18 controls (13 females, mean age = 12.2 years, SD = 1.8). All participants in the ADHD group and four participants in the control group had previously taken part in Experiment 1. The mean *T*-scores in the Conners-3 subscale of inattention were 74.4 (SD = 14.7) for the ADHD group and 52.3 (SD = 11.8) for the control group. The mean *T*-scores in the subscale of hyperactivity-impulsivity were 71.06 (SD = 19.3) in the ADHD group and 51.9 (SD = 9.7) in the control group. Annex 1 contains the complete characterization of participants in Experiment 2.

We ran Bayesian independent samples *T*-tests to verify whether the ADHD group and the control differed in the scores of inattention and hyperactivity-impulsivity. We set the alternative hypotheses as $T\text{-scores}_{(ADHD)} > T\text{-Scores}_{(controls)}$. We found very strong evidence of a group difference regarding the *T*-scores of inattention ($BF_{10} = 917.01$, error =

9.67 x 10^{-8}) and hyperactivity (BF_{10} = 70.91, error = 1.87 x 10^{-4}), confirming that clinical symptoms were present in participants diagnosed with ADHD and absent in controls.[18]

6.1.2. Material and stimuli

Our stimuli pool was formed by 46 pseudo-random series of consonants from the French alphabet, except for the letters "W" and "Y" as their French multisyllabic name is too long to utter. There was no repetition of letters within a series and we avoided acronyms. The series had increasing length from two to eight consonants, with three series of each length per pace condition, plus two extra series of two consonants for the practice trials in each pace condition. For the processing task, we created a set of 117 pseudo-random series of 12 digits and a set of 117 pseudo-random series of four digits (Hindo-Arabic numerals). We avoided obvious numerical sequences and too many repetitions of digits within a series (e.g., "12345", "7777").

The letters were displayed in font "Arial" (height set to 0.1 in Psychopy settings) and in dark red (colour name "maroon", RGB values: 128, 0, 0). The digits were displayed in the same font and size but in white colour. The central fixation point was a white asterisk (font "Arial", height set to 0.1 in PsychoPy settings). The background colour of the window was set to grey (RGB values: 175, 175, 175).

6.1.3. Procedure

Participants were presented with the same series of two to eight consonants, in increasing length. Each trial began with the display of the fixation point for 750 ms, followed by 500 ms of a blank screen and the display of one letter at the centre of the screen for 1500 ms. The presentation of the letters was interspaced by an interval of 10 seconds, during which a series of digits was successively displayed at the centre of the screen. After the presentation of the last letter of the series, the word "Rappel" ("recall" in French), was displayed at the centre of the screen and the participant was asked to orally recall the sequence of letters seen in the trial.

[18] As in Experiment 1, some participants in the control group had *T*-scores that suggested the presence of inattentive and hyperactive symptoms albeit the pattern of responses of their parents to the questionnaire seemed incongruent. We repeated the preliminary analysis done in Experiment 1 to compare the performance of controls in the normal range of *T*-scores to controls in the limit range. Again, we found no differences between them, so they were all grouped in the control group. The complete analysis is in Annex 5.

In the slow pace condition, four digits were displayed during the 10-second interval in-between letters. Each digit was displayed for 1,875 ms and they were interspaced by 625 ms of a blank screen, totalling four periods of 2,500ms with 75% of display and 25% of inter-digit delay. In the fast pace condition, 12 digits were displayed during the 10-second interval in-between letters. Each digit was displayed for 625 ms and they were interspaced by 208 ms of a blank screen, totalling four periods of 833 ms with 75% of display and 25% of inter-digit delay. The slow-pace condition corresponded to a ratio of 0.4 digit-per-second, and the fast pace condition corresponded to a ratio of 1.2 digit-per-second. Participants were required to read aloud all the letters and digits on trial and to keep the pace of reading during the task (Figure 8).

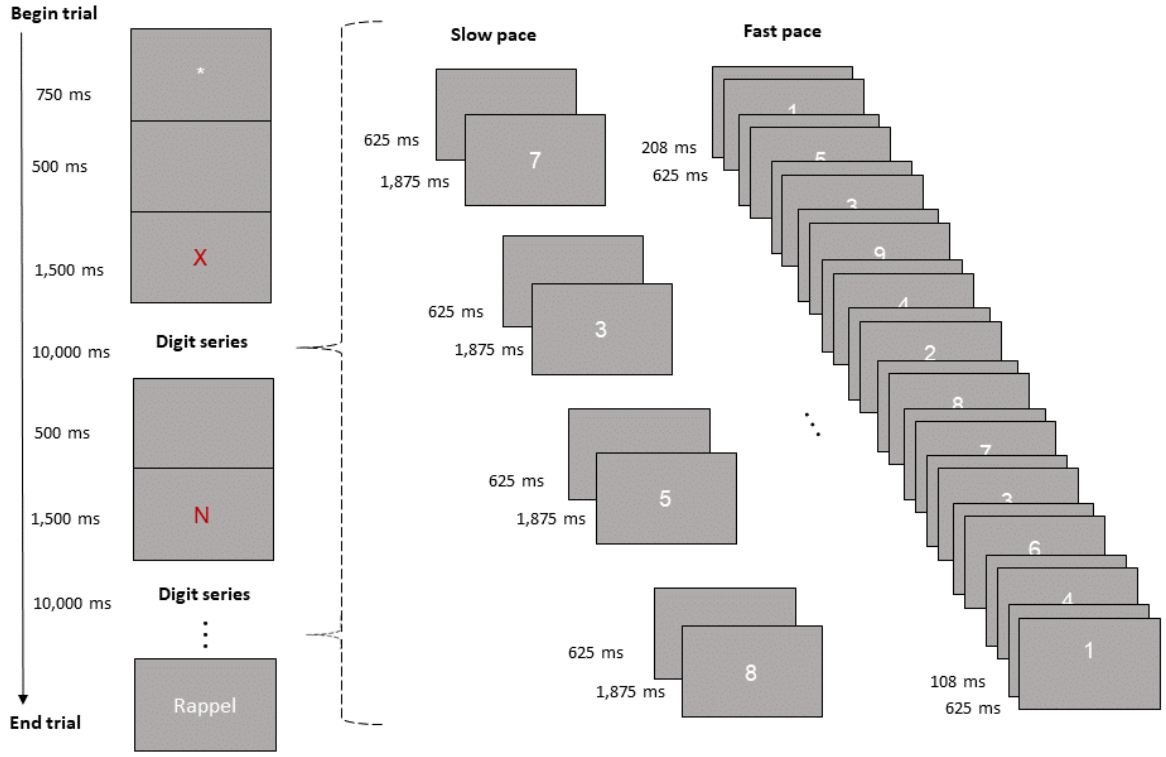

Figure 8. The reading digit span task. The length of the letter sequences presented in a trial increased at every three trials, starting with a sequence of two letters until the maximum length of the eight. The total interval between each letter of the sequence was kept constant (10,000 ms), but the pace of presentation of the digits during this interval varied. The size of the stimuli is not in scale in the figure.

The pace conditions were manipulated between blocks of trials and the order of presentation of the blocks was counterbalanced between participants. Each block began with a training of the pace of the reading digit task, with two practice trials containing only digits. Participants were instructed to read the digits aloud as they appeared on the screen and to maintain the pace of uttering during the whole task. After these two practice trials, the experimenter informed the participant that she must memorize a letter and an example trial with a single letter was given. After the participant's response, the sentences "*Mais c'est trop facile avec une seule lettre ! On essaye plus dur ?* " ("It's too easy with a single letter! Let's try harder?") appeared on the screen and the participant performed two practice trials of length two. After the training of the pace, and the example and practice trials, the experimental session began. Each block contained a maximum number of twenty-one trials, with three trials per sequence length (e.g., three trials of two-letter-sequences, three trials of three-letter-sequences, so on). After three trials of a particular level, a message warning the length of the following three series of letters was shown on screen. At the end of each trial, participants were required to recall aloud the sequence of letters seen on trial, in the same order that they were presented. The participants' responses were recorded by hand by the experimenter and no feedback was given during the task. The procedure ended when a participant failed to recall all three series of letters at a given length. All the response sheets were digitalized, and the answers were transferred to an Excel spreadsheet immediately after the experimental session.

6.1.4. Data analysis

As in Barrouillet et al. (2009), we attributed 1/3 to each correctly recalled series of letters and added the thirds plus one to calculate the span of each participant. The addition of one to the total number of thirds is required because the minimum length of series in the task is two. For example, someone who correctly recalled three series of two, two series of three, three series of four, and one series of five has a span of ((3+2+3+1) x 1/3) + 1 = 4.

We analysed the data considering the following dependent variables: the mean span and the percentage of letters recalled in correct serial positions. We calculated the percentage of letters recalled in correct positions (henceforth, percentage of letters recalled) for each participant considering the number of letters shown to her during the experimental session. The percentage of letters recalled is a measure that reflects a participant's span but

it contains additional information on the proportion of letters mistakenly recalled. While the span is a computation of full sequences correctly recalled (i.e., when all the letters are recalled in the correct serial position), the percentage measure reveals a proportion of how many letters within the sequences were correctly recalled, instead of a binary measure of correct versus incorrect sequences. For instance, one omission in a sequence of four letters (i.e., HBZK recalled as HZK, omission of the "B" in the original sequence) produces a smaller proportion of errors (25% of error) than a permutation of three letters in a sequence of four letters (i.e., HBZK recalled as BZHK, 75% of error). Yet, both responses exemplified are counted as one error in the calculation of the span. Therefore, the analysis of the percentage of correct letters can inform us about memory for items within the sequences, whereas the spans inform us about memory for the full sequences and the serial positions therein.

We ran Bayesian repeated-measures ANOVAs with the group (ADHD vs. control) as a between-subject factor and the pace condition (slow vs. fast) as a within-subject factor to compare the two variables across participants and conditions.

6.2. Results

6.2.1. Mean spans

In the ADHD group, the average mean span was 3.98 (SD = 1.24) in the slow pace and 2.54 (SD = 0.89) in the fast pace. In the control group, the average mean span was 4.23 (SD = 1.48) in the slow pace condition and 2.38 (SD = 0.80) in the fast pace condition. Figure 9 depicts these results.

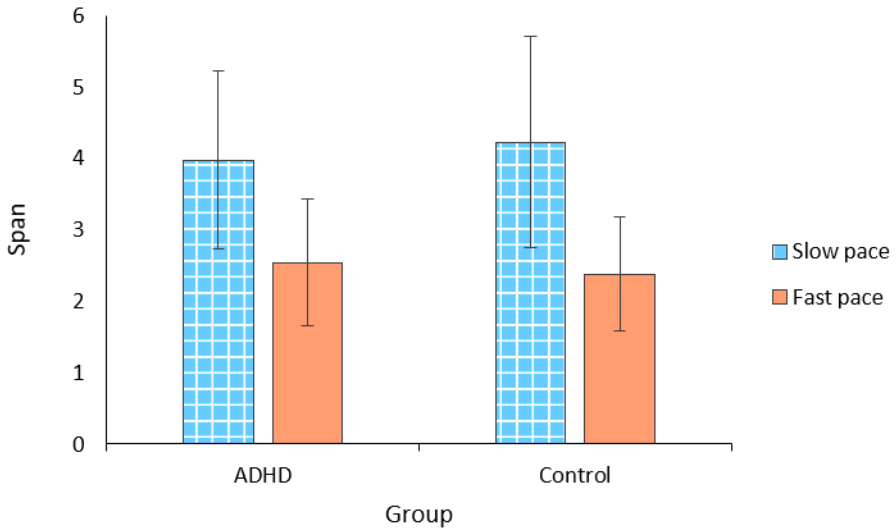

Figure 9. Mean spans per group and pace condition in Experiment 2. The vertical bars represent the standard deviations.

We compared the mean spans between groups and experimental conditions by running a Bayesian repeated-measures ANOVA. According to this analysis, the model that best fitted the data was the one considering only the main effect of the pace (BF_{10} = 1.773 x 10^7, error = 09). According to this model, recall performance was hampered in the fast pace both in the ADHD and control groups. The second-best model was the additive model including the main effects of the pace and the group (BF_{10} = 0.6751 x 10^7, error = 2.08), followed by the full model including the two main effects and the interaction between them (BF_{10} = 3.218 x 10^6, error = 2.55). Because the Bayes factors of the two best models did not differ strongly, we examined the BF_{incl} and BF_{excl} of each factor to better understand how they accounted for the data. We found extreme evidence for the inclusion of the pace effect (BF_{incl} = 1.423 x 10^7) in the model, but not for the exclusion of the group effect (BF_{excl} = 2.66) and the interaction between group and pace (BF_{excl} = 1.90).

The results of the Bayesian ANOVA showed a strong effect of the pace upon the mean spans and did not confirm our hypothesis predicting an interaction between group and pace. Indeed, the data pattern was essentially the same for ADHD and controls (Figure 9), despite the non-decisive evidence for the exclusion of the group effect and the interaction between group and pace in the model. To better account for a possible group effect, we ran pairwise sequential comparisons between the two groups in each pace condition using *T*-tests for independent samples. This type of analysis allowed us to

visualize a trend, if any, in the cumulative evidence in favour of the null hypothesis predicting no differences between the two groups. We set the alternative hypotheses as Mean span$_{(ADHD)}$ < Mean Span$_{(Control)}$ because we predicted that ADHD participants would have poorer recall performance than controls in both pace conditions.

The sequential Bayesian analysis revealed that ADHD participants were as performant as controls in both pace conditions. The cumulative Bayes factor fluctuated in the anecdotal range in the slow pace, whereas there was a clear trend towards moderate evidence for the null hypothesis in the fast pace (Figure 10). The sequential pairwise analysis supported the absence of a group effect and suggested that the intra-group variability among ADHD participants in the slow pace accounted for the strong Bayes factors of the models including the group in the Bayesian ANOVA.

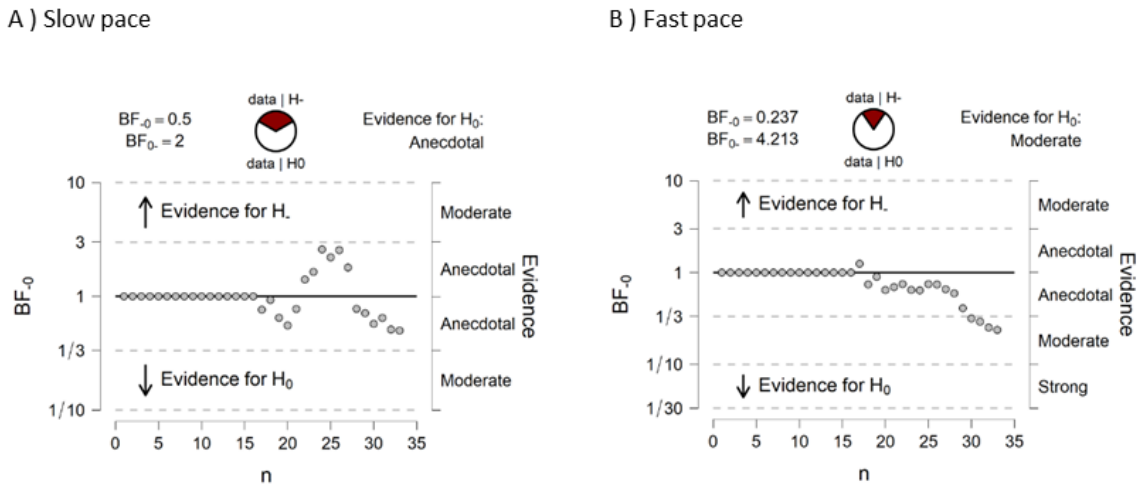

Figure 10. Sequential Bayesian analysis of group differences in spans at the slow (A) and fast (B) pace of Experiment 2. The statistical test used was a *T*-test for independent samples. The alternative hypothesis predicted lower spans in the ADHD group. In the two panels, the group of points to the right side of the image represent the sequential changes in the Bayes factor of the alternative hypothesis, at every addition of a data point corresponding to one ADHD participant.

The results presented so far support the interpretation that the fast pace disrupted performance in both groups and that ADHD participants were no less affected by it than the typically developing participants. We plotted the individual spans of the ADHD group

against the 95% confidence intervals of the controls in each experimental condition to better visualize the absence of a group difference (Figure 11).

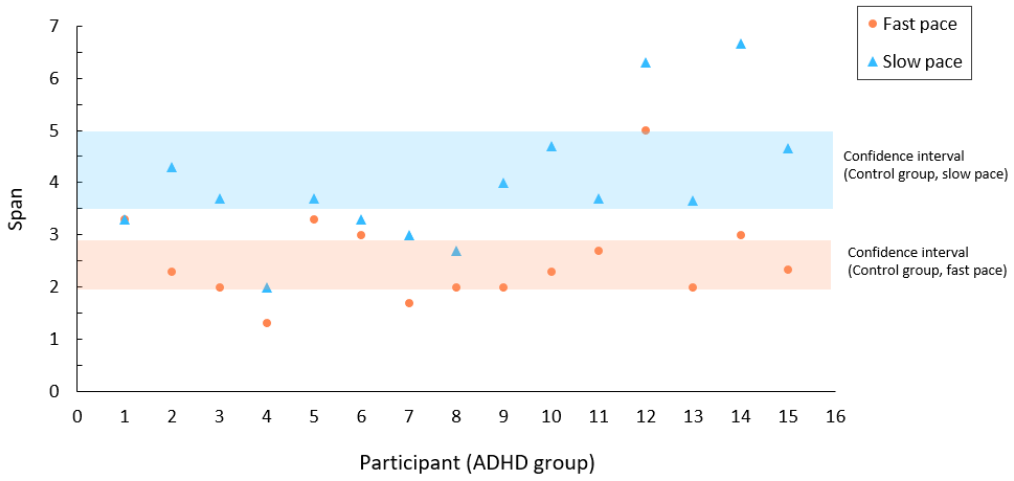

Figure 11. Individual spans of ADHD participants in Experiment 2. The blue zone represents the 95% confidence interval (3.50 – 4.97) of controls in the slow pace and the orange zone represents the confidence interval (1.98 – 2.78) in the fast pace.

The distribution of the data points shows that there was an overlap between the spans of the ADHD participants and the confidence intervals of the control group in the slow and fast pace, supporting the absence of the group effect and of the hypothesized interaction. In addition, the clear separation boundaries between the confidence intervals of the controls (blue and orange zones) and the spans of the ADHD in the fast pace (orange dots) consistently below the spans in the slow pace (blue dots) confirmed the strong effect of the pace manipulation. Except for one participant whose recall performance was unaffected by the pace manipulation (participant 1), all participants in the ADHD group were prone to the deleterious effect of the fast pace. There is, however, great variability on how much the fast reading of the digits hindered the serial recall of the letters in ADHD participants. For instance, while some participants were largely disrupted, with a performance loss between 3.7 to 2 letters (participants 2, 9, 10, 14, and 15), other had a performance drop of in only one letter, or even less (participants 4, 5, 6, 8). Interestingly, participants who were most disrupted by the fast pace were also the most performants in the slow pace (i.e., higher spans), and vice versa.

We hypothesized that the level of inattention and the age of participants modulated the loss in performance caused by an increase of the CL in the fast pace. It is possible that participants with higher levels of inattention were the less affected by the manipulation of the pace because their baseline performance in the slow pace was already poor, suggesting a difficulty in using attentional refreshing even when the processing component of the task allows plenty of free time to implement refreshing. Moreover, there is a developmental trend in the use of attentional refreshing, with children becoming more proficient in using it when they reach adolescence (Barrouillet et al., 2009) and therefore being more prone to pace manipulations that disrupt the availability of attention to refresh items in working memory. To account for these possible relationships, we ran a Bayesian repeated-measures ANOVA with the factors age, inattention and hyperactivity as covariates. Both ADHD and control participants were included in this analysis.

The model that best fitted the data was the one including the main effects of pace and age (BF_{10} = 1.635 x 10^8, error = 2.05). The second-best model was the one including the main effects of pace, age, and inattention, with a Bayes factor very close the best model (BF_{10} = 1.615 x 10^8, error = 1.36). We found decisive evidence for the inclusion of the pace effect (BF_{incl}= 4.346 x 10^7) and moderate evidence for the inclusion of the age effect (BF_{incl} = 8.66) in the model. Regarding the *T*-scores of inattention, the evidence in favour of their inclusion in the model was anedoctal (BF_{incl} = 1.293). The complete results with the Bayes factors of all models are in Annex 6. According to these results, the age of the participants accounted for most of the pace differences in their spans, with older participants being more affected by the fast pace than younger participants. Moreover, although the second-best model suggests that there was a small modulation of the symptomatic levels of inattention, the influence of age outweighed the influence of inattention upon recall performance.

To summarize, the set of results regarding the mean spans in Experiment 2 showed a strong effect of the pace manipulation and the absence of a group difference between ADHD and controls. The magnitude of the pace effect within the ADHD group was heterogeneous, with some participants being more disrupted by it than others. Finally, the analysis with the age and symptoms levels as covariates showed that the age of the participants accounted for most of the differences in their spans.

6.2.2. Percentage of letters correctly recalled

Overall, the analysis of the percentage of correct letters confirmed the results regarding the mean spans. On average, participants in the ADHD group recalled 74% (SD = 9%) of the letters in the slow pace condition and 62% (SD = 12%) in the fast pace condition. Participants in the control group, in their turn, recalled 73% (SD =13%) of the letters presented in the slow pace condition and 59% (SD = 13%) of the letters presented in the fast pace condition. Figure 12 shows these results.

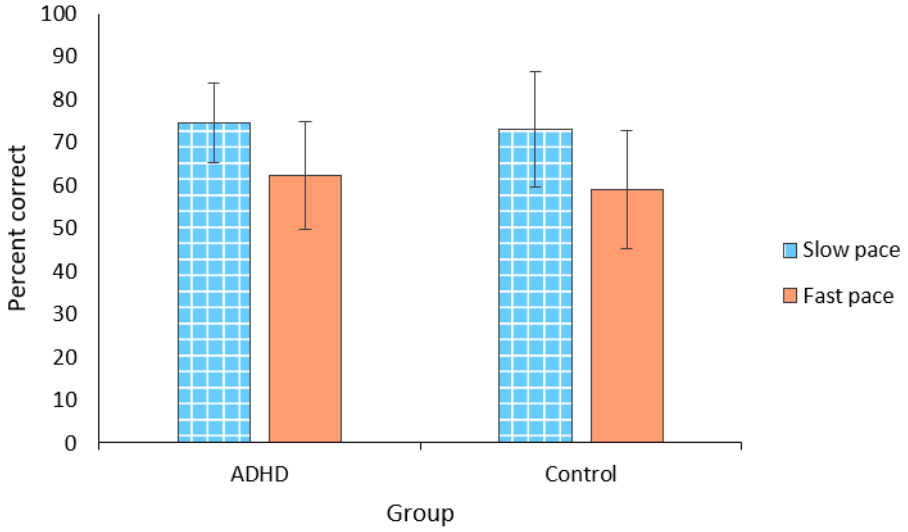

Figure 12. Percentage of letters correctly recalled per group and pace condition in Experiment 2. The vertical bars represent the standard deviations.

The Bayesian ANOVA revealed that the best model accounting for the data was the one including only the main effect of the pace manipulation (BF_{10} = 2.3314 x 10^4, error = 1.16), and the second-best model is the one including the main effects of the pace and the group (BF_{10} = 1.0171 x 10^4, error = 1.3), followed by the full model (BF_{10} = 0.3725 x 10^4, error = 3.09). Like for the mean spans, the examination of the BF_{incl} and BF_{excl} revealed very strong evidence for the inclusion of the pace effect (BF_{incl} = 1.808 x 10^4) in the model. Again, the evidence for the exclusion of the group effect (BF_{excl} = 2.51) and the interaction group x pace (BF_{excl} = 2.24) was not decisive, confirming the analysis of the mean spans.

We repeated the Bayesian sequential pairwise comparisons (*T*-tests) between the two groups in each pace condition, by setting the null hypotheses as $Percentage_{(ADHD)} < Percentage_{(Controls)}$. The sequential analysis revealed a clear trend towards moderate evidence for the null hypothesis in both pace conditions, with slightly more variation in the Bayes factor in the slow pace than in the fast pace (Figure 13).

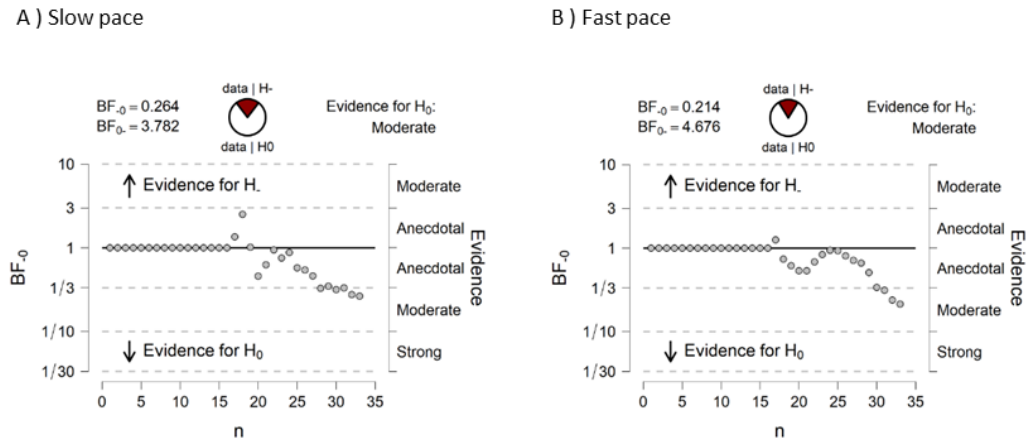

Figure 13. Sequential Bayesian analysis of group differences in the percentage of correct letters in the slow (A) and fast (B) pace. The statistical test used was a *T*-test for independent samples. The alternative hypothesis predicted lower percentages in the ADHD group. In the two panels, the group of points to the right side of the image represent the sequential changes in the Bayes factor of the alternative hypothesis, at every addition of a data point corresponding to one ADHD participant.

Both the Bayesian ANOVA and the sequential analysis of the percentage of correct letters confirmed the analysis of the mean span and reinforced our interpretation that the pace manipulation affected memory recall similarly across the groups of participants. Therefore, the analysis of the percentage of correct letters not only showed that the fast pace induced more errors at the sequence level (as indexed by their spans), but also at the item level (as indexed by smaller percentages), with no differences between the groups.

Again, the plot of individual data points in the ADHD group against the 95% confidence intervals of controls illustrates the effect of the pace and the absence of a group effect (Figure 14). The majority of ADHD participants (12 over 15) recalled a smaller proportion of letters in the fast pace, except one participant who was unaffected by it (participant 15) and two participants who recalled a greater proportion of letters in the fast pace (participants 1 and 5). The data on the percentage of letters were less heterogeneous

than the mean spans (compare to Figure 11), with a more consistent effect of the fast pace upon the percentage of correct letters than upon the mean spans. This suggests that, globally, the effect of the fast pace was less variable upon the proportion of letters recalled by ADHD participants than upon their spans.

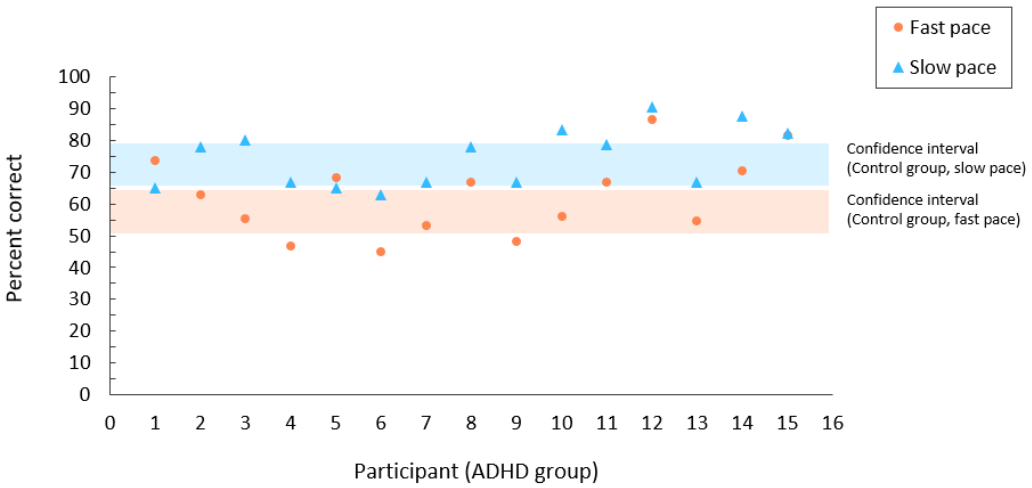

Figure. 14. Individual percentage of correct letters recalled by ADHD participants in Experiment 2. The blue zone represents the 95% confidence interval (66%-79%) of controls in the slow pace and the orange zone represents the 95% confidence interval (52% - 65%) of controls in the fast pace.

Finally, we ran a Bayesian repeated-measures ANOVA with age and *T*-scores of inattention and hyperactivity as covariates to account for the heterogeneity in our sample. We included both ADHD and controls in this analysis. The model that best fitted the data was the one including only the main effect of the pace (BF_{10} = 2.2908 x 10^4, error = 0.96), followed by the model including the main effects of the pace and the age (BF_{10} = 1.5958 x 10^4, error = 1.73). There was very strong evidence for the inclusion of the pace effect in the model (BF_{incl} = 2.1984 x 10^4) and the evidence for the inclusion of the age was anedoctal (BF_{incl} = 0.797). These results suggested that, differently than in the mean spans, the effect of the pace upon the percentage of correct letters was not modulated by the age of the participants.

6.3. Discussion

We designed Experiment 2 first to examine the group difference in the use of one particular attentional mechanism, refreshing. To this aim, we induced an increase of the cognitive load (CLs) of the task by varying the pace of presentation of the processing

component of a complex span task. The cognitive load effect indexes one's ability to use free time to use attention to refresh items, with higher CLs reducing the availability od attention and being more deleterious to its use. Studies in typically developing children showed that there is a developmental trend in the use of attentional refreshing that starts around age 6-7 and matures until adolescence (Barrouillet et al., 2009; Camos & Barrouillet, 2011). Therefore, we chose an age group of 10 to 16 years based on this trend. It was expected that participants in the control group would be proficient in using attentional refreshing and would be affected by the manipulation of the pace – as it was indeed observed in the results. Because children with ADHD have pervasive symptoms of inattention and/or hyperactivity throughout their development, we did not expect them to use attentional refreshing by the same age as the control group, or at least to have a less efficient use of it. Our hypothesis was contradicted by the results of Experiment 2.

The results of Experiment 2 showed a very strong effect of our experimental manipulation upon span and the proportion of correct letters recalled by participants. The deleterious effect of the fast pace upon performance reveals the presence of the cognitive load effect predicted by the TBRS model in our sample. Specifically, the observation of a pace effect in the ADHD group suggests that the working memory of children and adolescents in this clinical population is subject to the same temporal constraints that typically developing children and adults (Barrouillet et al., 2011; Camos et al., 2009; Gaillard et al., 2011). Moreover, the presence of the CL effect in ADHD participants suggests that this population is able to employ attentional refreshing to maintain active representations in working memory. To our knowledge, this is the first observation in the literature that the CL effect predicted by the TBRS model of working memory replicates in a sample of ADHD participants in a verbal serial recall task.

In a study by Weigard and Huang-Pollock (2017), the performance of ADHD children (ages between 8 and 10 years) was also affected by a manipulation that induced changes in the CL in a complex span task tapping the visuospatial domain of working memory. In their study, the task consisted in memorizing spatial sequences (i.e., squares presented in a spatial array) and making numerosity discriminations (i.e., respond whether a grid contained "a lot" or "a little bit" of asterisks) between each memory item of the sequence. To manipulate the CL of participants, the authors induced slowed processing speeds in the numerosity task by varying the difficulty of the numerosity judgements. The authors varied the number of asterisks displayed to make the response boundary between

"a lot" and "a little bit" easy to discriminate in one condition and hard in the other. The results showed that processing speed was indeed slowed in trials with hard numerosity judgements condition, which caused an increase of the CL and its consequent decrement in the percentage of items recalled. More importantly, the causal relationship between slower processing speed, CL, and performance was observed both in controls and ADHD participants. The authors did not find an interaction between the speed condition and the diagnostic status of participants, a result that is consistent with our observations in Experiment 2, in which the hypothesized pace x group interaction was not found. The absence of these interactions suggests that the working memory capacity of children with ADHD was similarly affected by changes in the CL in both studies.

There is nevertheless one important difference between our results and the ones reported by Weigard and Huang-Pollock (2017). In their study, ADHD participants significantly performed poorer than controls in all experimental conditions, whereas we found no group differences in their mean spans and percentage of recalled letters. We believe that differences in the task modality and the sample characterization of the two studies might have played a role in this difference. First, we used a verbal complex span task whereas Weigard and Huang-Pollock used a visuospatial task. The working memory deficits in ADHD are more pronounced in the visuospatial domain, as documented in meta-analyses including neuropsychological and experimental studies in the ADHD population (Martinussen et al., 2005; Kasper et al, 2012; Willcutt et al., 2005). Hence, it is possible that our task was less sensitive to their difficulties compared to typically developing controls. Through pure observation, we noticed that our ADHD participants attempted to use overt phonological rehearsal between the digits in the sequence, a strategy that could compensate their deficits and mask the differences between the two groups in our Experiment 2. Weigard and Huang-Pollock (2017), in their turn, used a spatial task that makes phonological rehearsal of the memory items more difficult. Therefore, it is possible that their participants were more reliant on attention-based strategies.

Second, our sample included older participants (mean age = 13.06 years compared to the 10.11 years of age for Weigard and Huang-Pollock) that can account for higher span and percentage of recalled letters, bringing the mean closer to controls. Regarding the clinical characterization of participants, Weigard and Huang-Pollock (2017) did not report the diagnostic subtype of participants (inattentive, hyperactive, or combined), but the reported *T*-scores of inattention (mean = 68.99) and hyperactivity-impulsivity (68.00) in

the Conners-3 Parent scale are slightly below the ones we found in our sample (74.40 for inattention and 71.06). Regardless of the differences in the *T*-scores between the two samples, the results reported by Weigard and Huang-Pollock (2017) and in our Experiment 2 suggest that the CL effect was not modulated by the presence of inattentive symptoms. In their case, the absence of an interaction between speed condition and diagnostic status is suggestive of this conclusion. In ours, it is the absence of the interaction between pace and group and the negligible effect of the inattention as a covariate in the model.

In addition to the strong effect of the pace manipulation in our Experiment 2, the Bayesian ANOVA considering the age and the symptoms levels as covariates also revealed a strong effect of the age upon the mean spans. This result suggests that, at least in our sample, the use of attentional refreshing by ADHD participants followed the same developmental trend observed in typically developing children reported in the literature. According to this analysis, the use of attentional refreshing is optimized in older participants in our sample, as revealed by an increasing impact of the CL with the age of participants. It is worth noting that the psychiatric literature has well documented that the inattentive symptoms tend to augment at the beginning of adolescence of children diagnosed with ADHD. This trend in the prognostic of ADHD goes in the opposite direction of a modulation effect of age upon the use of attentional refreshing. If children diagnosed with ADHD become more inattentive when entering adolescence, why would we observe an improvement in the use of attentional refreshing in working memory? Our tentative answer to the question is that, despite their baseline attentional deficit compared to typically developing children, ADHD children optimize their use of attentional refreshing according to the available attentional resources.

The results from Experiment 2 do not allow extrapolations regarding the magnitude of this developmental trend in ADHD children compared to controls, but they offer a first indication that a similar pattern of growing use of attentional resources in working memory could in ADHD. A previous study, Barrouillet et al. (2009) used the same complex span task than in Experiment 2, i.e., the reading digit span task, to assess the effects of the pace on recall memory across a large age range. The slow and fast pace conditions in our Experiment 2 were based on two conditions in this study, which allowed us to make comparisons between their findings and the current results.

For a similar age group (i.e., the 12-year-olds) of typically developing children and the same rates of digits presentation, Barrouillet et al. (2009) reported a mean span of around 5 letters in the condition at which digits were presented at a pace of 0.4 digit/s (i.e., a pace similar to our slow pace) and 3.3 letters when digits were presented at 1.2 digit/s pace (similar to our fast pace). In Expriement 2, we observed a mean span of 4.2 and 2.3 letters in our control group at the slow and fast pace, respectively. In our ADHD group, we found a mean span of 3.9 at the slow pace and 2.5 letters at the fast pace. Participants in the study by Barrouillet et al. (2009) outperformed our participants in the reading digit span task in about one letter, both ADHD and controls. Nevertheless, the drop in performance caused by the fast pace was similar across participants in the two studies: about 1.7 letters in Barrouillet et al. (2009), 1.7 in our control group, and 1.4 in our ADHD group. Although it is difficult to make direct comparisons between the two studies, our results suggest that, at least in our sample, ADHD were subject to a CL effect similar in magnitude to a previous report in the literature for the same age group of typicalling developing individuals.

Given the well-documented developmental trend in the use of attentional refreshing in typically developing children, we believe that the similar magnitude of the CLs between our study and Barrouillet et al. (2009) offers a first clue that this strategy can develop with age also in ADHD. To end, the results of Experiment 2 encourage future studies to further investigate the optimization of attention-based maintenance mechanisms in working memory in the population of children with ADHD. In the next chapter, we will present an experiment designed to test the opposite prediction of Experiment 2, i.e., whether a pace manipulation enabling more free time to use refreshing will alleviate the CL and consequently cause an improvement in recall memory.

Chapter 7

Experiment 3: The adapted reading span task

Experiment 3 consisted in a complex span task tapping the verbal and visuospatial domains of WM. The paradigm and rationale behind Experiment 3 are very much alike to Experiment 2, but instead of requiring participants to read digits aloud between the memory items (i.e., the letters), the task in Experiment 3 required them to perform spatial judgements, henceforth called "spatial fit". The spatial fit consisted in judging whether a horizontal bar fits the gap between two squares.

There are two fundamental differences between experiments 2 and 3. First, Experiment 2 mainly disrupts the phonological loop of WM during the task, as it is occupied by the reading of the digits in-between the memory items. Thus, the use of articulatory rehearsal to maintain the letters is hampered. Experiment 3, in its turn, mainly disrupts the use of attention during the maintenance of items, as the spatial fit requires participants to deploy attention to judge the size of the bars. Second, the paces of presentation of stimuli (1.2 digit/s and 0.4 digit/s) in Experiment 2 were predetermined and kept constants for all participants – that is, every participant performed the task at the same pace as the others. In Experiment 3, we manipulated the pace of one block of trials by adapting it to each participant's mean RT during the spatial fit. Therefore, participants performed the task at a predetermined pace (control condition) and at an adapted pace (tailored according to their individual mean RT).

It is known that children with ADHD have slower processing speed than their typically developing peers, as assessed by slower and more variable RTs (Castellanos et al., 2005; Metin et al., 2012). Meta-analytical and experimental studies revealed that the effect sizes of ADHD upon RT speed and variability ranges from moderate to large (Karalunas et al., 2014; Kofler et al., 2013) and that increased RT variability is a specific marker of ADHD compared to other psychopathologies with overlapping symptomatology (e.g., oppositional defiant disorder, conduct disorder, autism spectrum disorder) (Salum et al.,

2019; Karalunas et al., 2014). For these reasons, slower processing speed in the ADHD group can partially account for residual group differences in Experiment 2 after manipulating the CL by varying the pace of the reading of the digits.

As explained in the introduction of this thesis (Chapter 1, section 1.2.), the TBRS model defines the cognitive load (CL) as the ratio between the time that attention is occupied by the concurrent processing task (*Ta*) and the total time available to perform it (*Tt*), that is, CL = *Ta*/ *Tt*. *Ta* is not the same for all participants: if a participant has a faster processing speed of stimuli (i.e., faster RTs in the concurrent task), then *Ta* is lower and so will be the CL. We also saw that, according to the TBRS, the higher is the CL, the lower is the performance in terms of mean spans. From these two postulates we can derive that:

1) If there are individual differences between participants regarding their processing speed, providing the same time to perform the concurrent task (*Tt*) will not necessarily make all participants perform the task under the same CL. Otherwise stated: the same pace of presentation of stimuli does not yield the same CL to all participants.
2) To provide all participants with the same CL, we must tailor *Tt* to their personal *Ta.*

A similar approach has been taken by Weigard and Huang-Pollock (2017), who used a drift diffusion model to manipulate the processing speed of participants during a complex span task. They showed that, by slowing one's processing speed during the task, an increase in the CL predicted poorer WM performance for both ADHD and controls. In Experiment 3, we used each participant's processing speed to equalize the CL between ADHD and controls and test the hypothesis that an equal CL minimizes group differences in WM performance. This hypothesis has already been tested in typically developing children of different age groups by Gaillard et al. (2011). They showed that tailoring the pace of presentation of stimuli according to each participants' processing speed equalized the CL between the different age groups and abolished group differences in performance.

Experiment 3 is, therefore, a replication of Experiment 2 with an extra control variable: the CL. We manipulated the CL by tailoring *Tt* according to each participant's mean RT in the spatial fit task (an index of *Ta*). In the control condition, the participants had the same interval (*Tt*) to perform the spatial fit judgements, which implies that individual differences in *Ta* between ADHD and controls yield different CLs between the

groups. In the adapted condition, *Tt* was adjusted to the mean RT in the spatial fit, thus equalizing the CL between participants and groups. Therefore, Experiment 3 had a control condition in which the CL was different between groups and an adapted condition in which the CL was the same between groups.

We designed the three experiments in this thesis concomitantly in order to cope with time constraints related to the recruitment of participants. For this reason, the data collection of Experiment 2 was not yet completed when we designed our experimental reasoning and predictions for Experiment 3, thus we kept the original hypothesis of a group difference between ADHD and controls. Our predictions for Experiment 3 were: 1) memory performance would be greater in the adapted condition than in the control condition for ADHD participants; 2) the differences in memory performance between ADHD and controls would diminish or disappear in the adapted condition. If group differences disappear in the adapted condition, we could conclude that ADHD children and adolescents can perform attentional refreshing but need more time to do it properly. In this case, their worse WM performance in daily life activities can be attributed to time constraints on how tasks are built, but not necessarily to a lack of ability in putting attention-based maintenance into action. If group differences are only partially gone after controlling for the CL, we could conclude that ADHD children can refresh items in WM to some extent; nevertheless, they are subject to a more pervasive deficit in WM that our experimental manipulation cannot pinpoint.

Table 4 summarizes the differences in design between Experiment 2 and Experiment 3 and their respective hypotheses and predictions.

Table 4

Summary of the differences and similarities between Experiments 2 and 3.

	Experiment 2 The reading digit span task	Experiment 3 The adapted reading span task
Memory task	To recall letter series of increasing length (2 to 8 letters).	
Processing task	To read digits aloud.	To make spatial judgements.
Response modality	Serial recall (oral responses).	Serial recall (oral responses) and mouse clicks for the spatial fit.
Pace conditions	Slow pace (0.4 digits/s) Fast pace (1.2 digits/s)	Control pace (predetermined, 1,500 ms per spatial judgement) Adapted pace (1.5 x mean RT per spatial judgement)
CL conditions	Lower CL (slow pace) Higher CL (fast pace)	Variable CL per participant (control pace). CL = RT/1,500ms. $CL_{(ADHD)} \neq CL_{(control)}$ Same CL per participant (adapted pace) CL = RT/1.5x RT) $CL_{(ADHD)} = CL_{(control)}$
Predictions	If ADHD children can perform attentional refreshing, memory performance will be higher in the low CL condition in both groups.	If ADHD deficit in WM results from slower attentional refreshing, their memory performance should be closer to controls when the same CL is warranted.

7.1. Method

7.1.1. Participants

Fourteen participants took part in the ADHD group (3 females, mean age = 13.7 years, SD = 1.7) and 17 participants took part in the control group (9 females, mean age = 12.2 years, SD = 1.4). All participants in the ADHD group and one participant in the control group had taken part in Experiments 1 and 2. After the exclusion of two outliers in the first percentile, the control group included 15 participants (7 females, mean age = 12.3 years, SD = 1.4). Annex 1 contains the complete characterization o the sample in Experiment 3.

The mean *T*-scores of inattention in the Conners-3 subscale were 73.3 (SD = 14.6) in the ADHD group and 51.4 (SD = 6.3) in the control group. The mean *T*-scores of hyperactivity-impulsivity were 69.7 (SD = 19.3) in the ADHD group and 54.6 (SD = 7.3) in the control group. As in the two previous experiments, we tested whether the *T*-scores of inattention and hyperactivity-impulsivity were different between the two groups of participants. Again, we used Bayesian independent samples *T*-tests and set the alternative hypotheses as $T\text{-scores}_{(ADHD)} > T\text{-Scores}_{(controls)}$. We found very strong evidence of a group difference regarding the *T*-scores of inattention ($BF_{10} = 1141.8$) and moderate evidence of a group difference in the *T*-scores of hyperactivity ($BF_{10} = 5.66$), confirming that the two groups differed in terms of the presence of clinical symptoms.

7.1.2. Material and stimuli

We used the same stimuli pool from Experiment 2 for the letter sequences. For the spatial fit, we created 96 images of a horizontal bar flanked by two horizontally aligned squares (sides measuring 0.5 cm). The width of the horizontal bar was fixed to 0.5 cm and its length bar varied from 1.1 to 5.2 cm, and the gap between the two squares varied from 0.5 to 5.4 cm. The horizontal bar was located either above or below the two squares, and the vertical distance between the bar and the squares varied from 0.5 to 1.7 cm. The horizontal bar fitted the gap between the squares in half of the stimuli. A Bluetooth mouse was connected to the laptop for response collection in the spatial fit.

7.1.3. Procedure

The procedure of Experiment 3 followed the one of Experiment 2, except that the digits between the letters were replaced by four spatial fit judgements. In each trial, participants were presented with the same series of two to eight consonants, in increasing length. The temporal parameters for the display of the letters were the same as in Experiment 2, that is, 750ms of a central fixation point, followed by 500 ms of a blank screen and the display of one letter for 1500 ms. The presentation of the letters was interspaced by four spatial fit stimuli, sequentially presented at the centre of the screen. Participants should respond via mouse clicks if the horizontal bar fitted the gap between the two squares (right button for "Yes" and left button for "No"). We put coloured stickers (green for "Yes" and red for "No") on the mouse buttons to remind participants of their meaning. After the presentation of the last letter of the series, the word "Rappel" ("recall"

in French), was displayed on the screen and the participant was asked to orally recall the sequence of letters seen in the trial. The release of the next trial was controlled by the participant via mouse click. As in Experiment 2, the length of the letter series increased at every three trials and a motivation message saying "*Top! … lettres*" ("Top! … letters") with the length of the next series was displayed before each new level. The task was interrupted when the participant made three mistakes of the same length.

We manipulated the cognitive load of the task by varying the pace of presentation of the spatial fit in-between the letters. In the control condition, the participants had 1500ms to respond to each spatial fit. Each spatial fit was preceded by 500 ms of a blank screen and the stimulus was displayed for 1500ms at the centre of the screen. In the adapted condition, the time of presentation of the spatial fit was tailored according to their mean RT, calculated in advance during an assessment stage at the beginning of the experimental session. Each spatial fit was preceded by 500 ms of a blank screen and the stimulus was displayed for 1.5x(mean RT) ms. Response collection via mouse click was enabled during the display of the spatial fit and mouse clicks did not prompt the next stimulus. After a sequence of four spatial fit judgements, the next letter in the series was displayed. Figure 15 shows the procedure of Experiment 3.

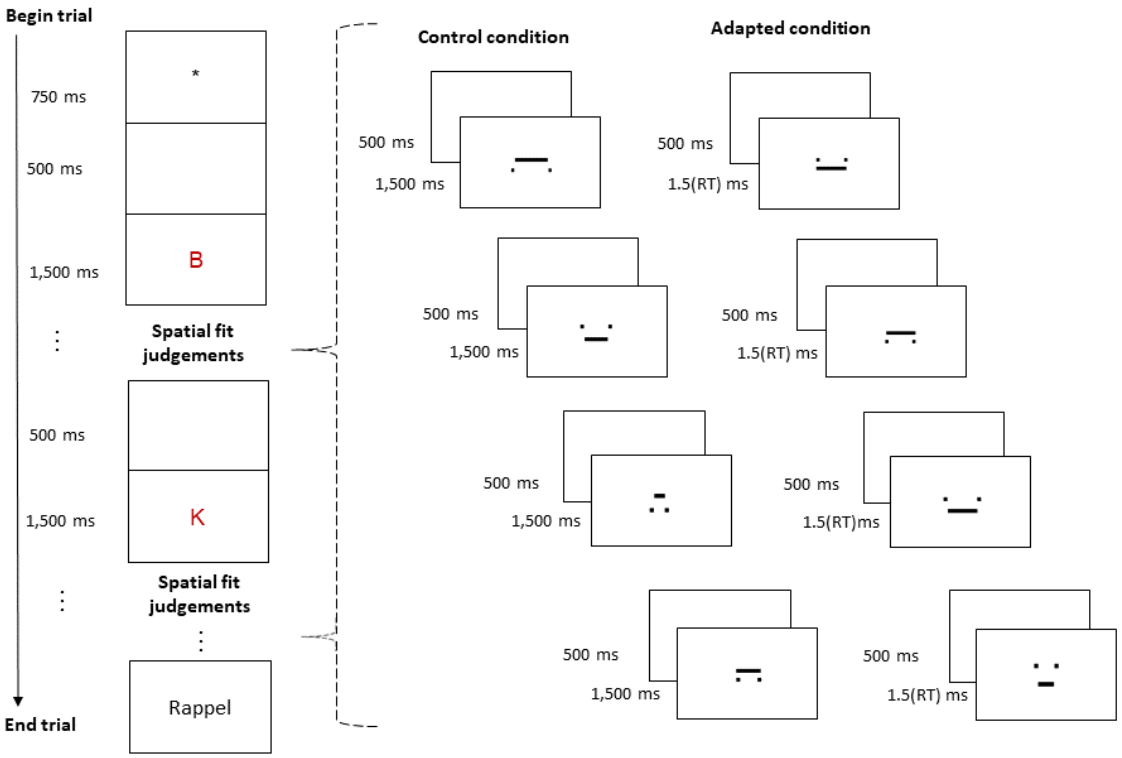

Figure 15. The adapted reading span task. The figure exemplifies the sequence of events in a trial of length 2 in Experiment 3. The length of the letter series increased every three trials. The three dots above the final screen "*Rappel*" (Recall) mean that this sequence of events was repeated in trials with longer series. Participants made four spatial fit judgements between each letter in a series. Stimuli are not depicted in scale in the figure.

The experimental session began with a stage of RT assessment in the spatial fit task, in which the program calculated the mean RT of the participant during the spatial fit. Participants were orally instructed that the goal of the task was to maintain letters and perform spatial judgements concomitantly, without neglecting any. The experimenter then showed two examples of spatial fit judgements and how to respond via mouse clicks. Following, she announced that the task would begin with a "challenge": the participant should score 10 points (correct responses) in the spatial fit before proceeding to the second stage of the task, involving the letters. A score was displayed on the upper right corner of the screen in this stage, and the participant made as many spatial judgements as needed until he/she reached 10 points. In this stage, the spatial fit stimulus was displayed for 1000 ms and followed by a blank screen. There was no time limit for responding and mouse responses prompted the onset of the next spatial fit. In case participants responded faster

than 1000 ms, the mouse clicks prompted the offset of the stimulus and the immediate onset of the next spatial fit (no blank screen interval). The program automatically calculated the participant's mean RT in correct trials by the end of the RT assessment phase; this was later used as a temporal parameter in the adapted pace condition. From this point, the score in the upper right corner was not exhibited anymore.

By the end of the RT assessment phase, the experimenter explained the complete task, and the participant performed two practice trials. The first practice trial had a series of one letter; the second practice trial had a series of two letters. The spatial fit stimuli were displayed for 1000 ms and there was no time limit to respond to it during the practice trials. After the practice trials, the experimenter explained that the task would become "harder" as the spatial fit would happen in a fixed time: if the participant does not respond on time, she will score zero; if she responds before the stimulus offset, she must wait until the next stimulus is displayed. Then, the participant performed one practice trial of length 2 with the fixed time parameters and the block of trials began.

The cognitive load was manipulated between blocks of trials and the order of presentation of blocks was counterbalanced between participants. When the stop rule of three consecutive errors of the same length was reached in the first block of trials, the experimenter prompted the program to proceed to the next condition block. At this point, the participant was simply told that the pace of the spatial fit was "going to change" and performed one practice trial of length 2 in the new pace condition. A motivation message saying "*Bravo! T'es arrivé.e jusqu'à … letters!*" ("Bravo! You made it to … letters!) with the longest letter sequence reached by the participant was displayed at the end of each block. Participants in the ADHD group were allowed small breaks during the task, usually 10 minutes between each block condition.

Figure 16 shows the execution flow of Experiment 3 and the order of events during the experimental session.

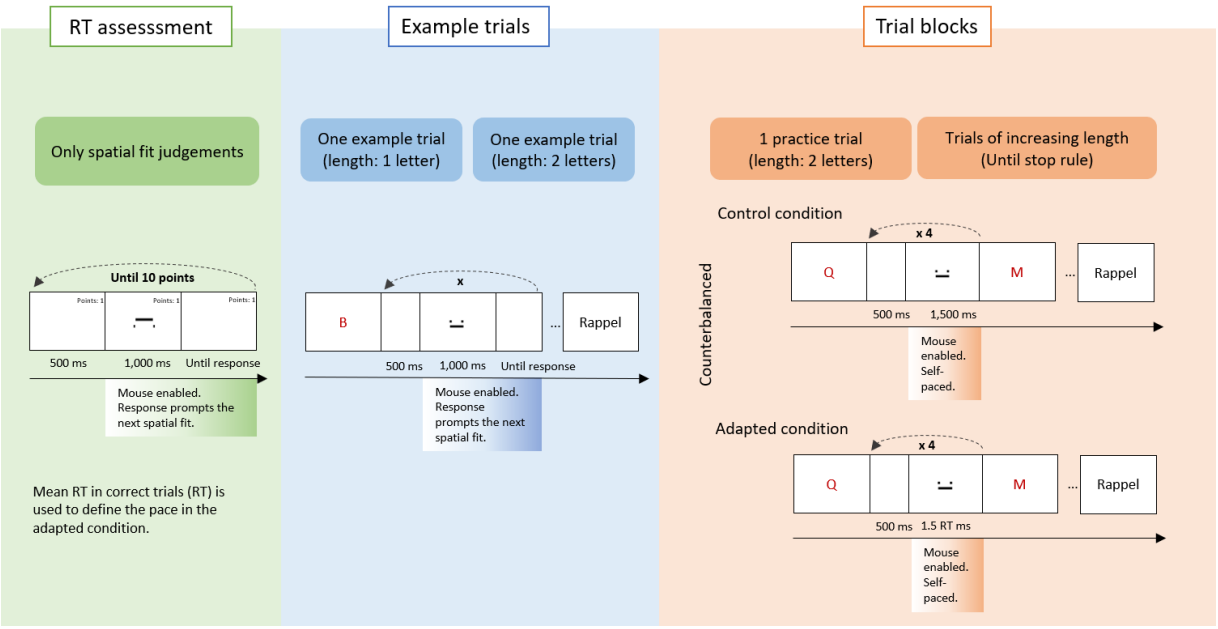

Figure 16. Schematic representation of the execution flow of Experiment 3. The inferior part of the figure details how response collection was implemented in the spatial fit task in different stages of the experiment. The stimuli are not depicted in scale.

7.1.4. Data analysis

We calculated each participants' span and percentage of letters recalled in correct positions. For the computation of the spans, we used the same formula proposed by Barrpuillet et al. (2009) and applied to data in Experiment 2. We compared the means of the two dependent variables across participants and pace conditions by using Bayesian repeated-measure $ANOVA_S$ with the group as a between-factor and the pace as a within-factor.

7.2. Results

7.2.1. Mean spans

In the ADHD group, the mean span was 4.3 (SD= 1.1) in the normal pace condition and 4.8 (SD = 1.06) in the adapted pace condition. In the control group, the mean span was 4.7 (SD = 0.9) in the normal pace condition and 4.7 (SD = 1.1) in the adapted pace condition. The results of the Bayesian ANOVA showed no evidence in favour of a pace effect (BF_{10} = 0.422, error = 0.81), a group effect (BF_{10} = 0.35, error = 1.12), nor their interaction (BF_{10} = 0.83, error = 1.62), therefore the best model accounting for the data was the null. Accordingly, we found moderate evidence for the exclusion of the factors pace

(BF_{excl} = 3.10), group (BF_{excl} = 3.59) and their interaction (BF_{excl} = 5.84) from the model (Figure 17).

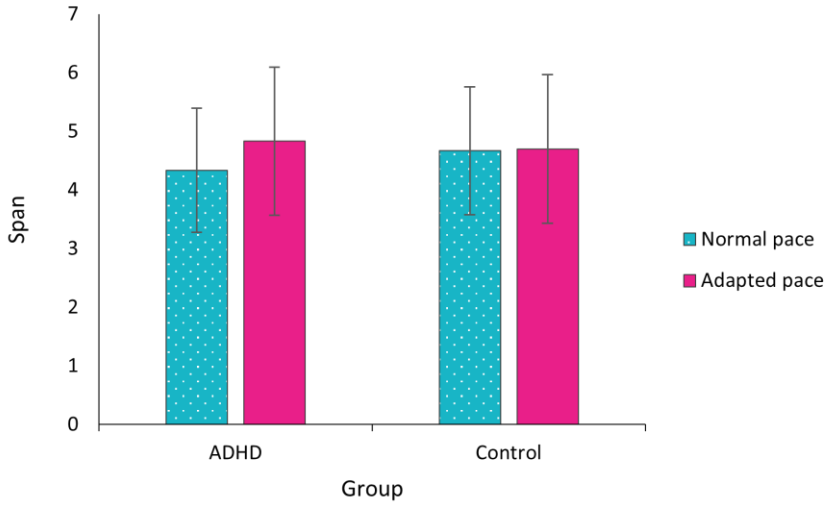

Figure 17. Mean spans per group and pace conditions of Experiment 3. The vertical bars represent the standard errors.

Following, we ran sequential Bayesian analyses comparing the two groups across pace conditions with independent samples *T*-tests to examine whether there was a consistent trend in the posterior odds towards the null. For this analysis, we set the alternative hypothesis as Mean $span_{(ADHD)}$ < Mean $span_{(Controls)}$, as we expected ADHD participants to have poorer performance. Figure 18 shows the results of this analysis in each pace condition.

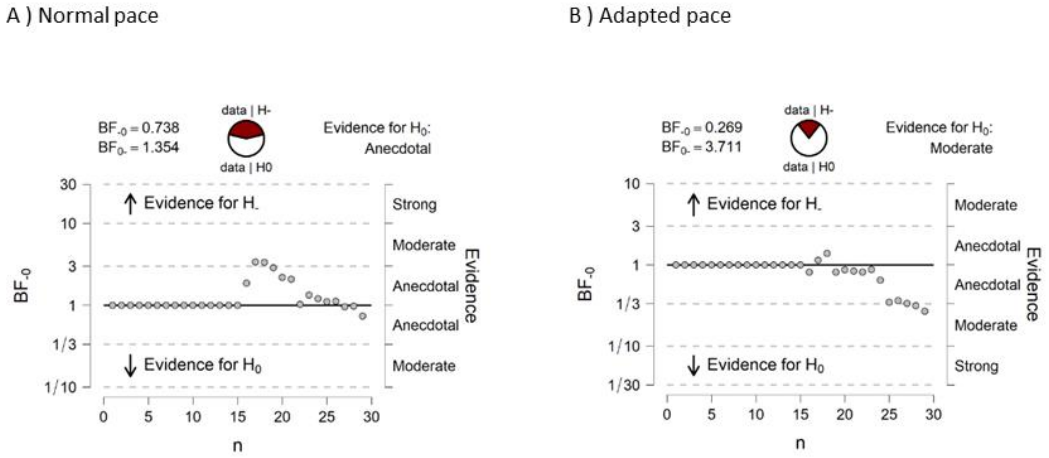

Figure 18. Sequential Bayesian analysis comparing the spans between the groups in the normal (A) and adapted (B) pace. The alternative hypothesis was $Span_{(ADHD)} < Span_{(Control)}$. In both panels A and B, the points to the right side of the figure represent the sequential changes in the BF_{10} of the model predicting lower spans in the ADHD group, at every addition of a data point corresponding to a span of an ADHD participant.

The examination of the sequential analyses showed that the initial moderate evidence (BF_{10} around 3 in data point 17) in favour of the alternative hypothesis shifted towards cumulative anecdotal evidence at the normal pace. In the adapted pace, there was a clearer trend in the cumulative Bayes factor towards moderate evidence in favour of the null. Together with the Bayes factors of the models presented above, the interpretation of the sequential analysis suggested that memory recall did not differ between the two groups of participants, nor it was lower in the ADHD group, as we expected.

A closer examination of the individual spans showed that performance was very heterogenous in the ADHD group and, differently from Experiment 2, no trend of a pace effect was detected. The plot of individual spans against the 95% confidence intervals of the means in the control group (Figure 19) helps us to visualize the heterogeneity in performance and the different impacts of the pace manipulation upon the spans of the ADHD group.

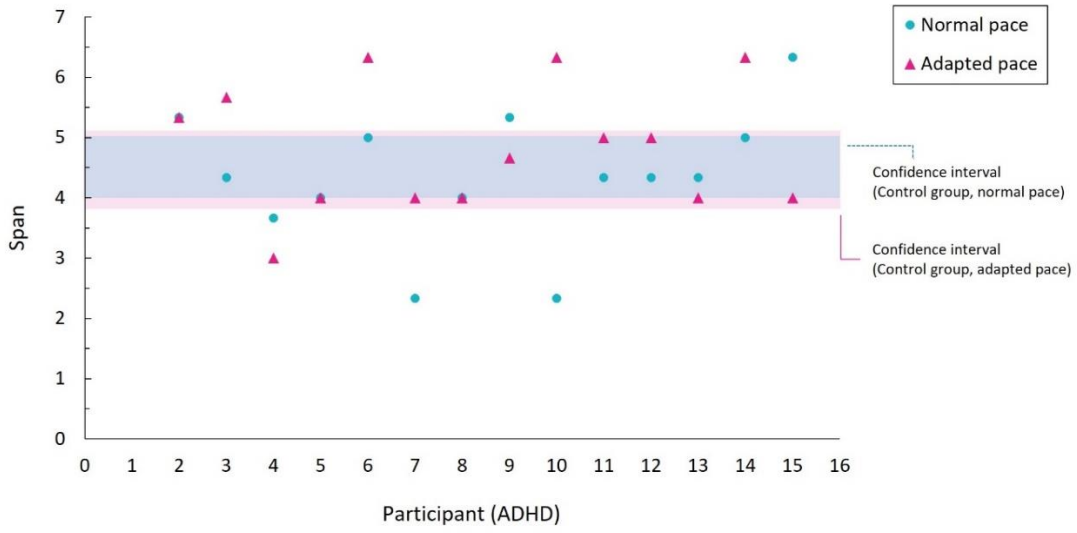

Figure 19. Individual spans in the ADHD group plotted against the 95% confidence intervals of the means in the control group, in each pace condition of Experiment 3. The shaded zone in blue represents the confidence interval for controls in the normal pace (4.17 - 5.16) and the shaded zone in pink represents the confidence interval for controls in the adapted pace (4.05 – 5.28).

As observed in Figure 19, there was large overlap between the spans in the ADHD group and the confidence intervals of controls. Moreover, it was impossible to detect a pattern on how the adapted pace affected the span of participants in the ADHD group: some participants were very sensitive to our manipulation of the pace and greatly benefited from the adapted pace (e.g., participant 10), whereas others were not affected (e.g., participants 2 and 5) or even disrupted by it (e.g., participants, 4, 9, and 15).

We designed Experiment 3 concomitantly to Experiment 2, so we were unaware of the absence of a group effect before we established our predictions. According to our initial hypothesis and previous findings in the literature reporting that ADHD exhibit a WM deficit (Kasper et al., 2012), data points in the ADHD group in Experiment 3 would have fallen below the confidence interval of controls in the normal pace and inside the confidence interval in the adapted pace (or closer to the lower boundary). Only the data points of one participant group (participant 7) behaved as we predicted. Instead, some ADHD participants behaved exactly as controls (participants 11 and 12), with spans falling into the confidence intervals in both conditions; some of them even overperformed controls (participants 6 and 14); and one participant who was nearly two letters behind controls managed to surpass them in one letter in the adapted condition, with a gain in performance

of 4 letters (participant 10). We believe that our heterogeneous sample regarding the age and the clinical subtypes of ADHD may have impacted the findings observed in Experiment 3, as discussed in the two precedent chapters.

To better account for individual differences in the levels of symptoms, we ran a Bayesian repeated-measure ANOVA with the *T*-scores of inattention and hyperactivity-impulsivity as covariates and we calculated their correlations with the spans in each pace condition. Congruent with the main ANOVA described above, the null model was the best one accounting for the data. There was no evidence of a correlation between the *T*-scores and the spans (see Annex 6 for the complete results). The second-best model in the ANOVA was the one including the main effect of the pace, but the evidence in favour of this model was negligible ($BF_{10} = 0.428$, error = 1.42). Regarding the Bayesian correlations, the evidence of a negative correlation between inattention, hyperactivity and the mean spans was negligible for all pairs of variables (all Bayes factors smaller than 1). We only found moderate evidence ($BF_{10} = 4.5$) of a moderate correlation (0.458) between the *T*-scores of inattention and hyperactivity, which confirms that the symptomatic presentation of our sample is similar to previous reports in the literature.

7.2.2. Percentage of letters correctly recalled

As in Experiment 2, we calculated the percentage of letters recalled in correct positions for each participant in each experimental condition. This analysis essentially confirmed the analysis of the mean span. On average, participants in the ADHD group recalled 72.3% (SD= 8.7%) of the letters in correct positions in the normal pace condition and 72.1% (SD = 12.1%) in the adapted pace condition. In the control group, the average percentage of correctly recalled letters was 74.6% (SD= 6.8%) in the normal pace condition and 73.1% (SD = 4.9%) in the adapted pace condition. Also, the data distribution was very heterogeneous in the ADHD group, overlapping with the confidence intervals of the control group. Figure 20 shows these results.

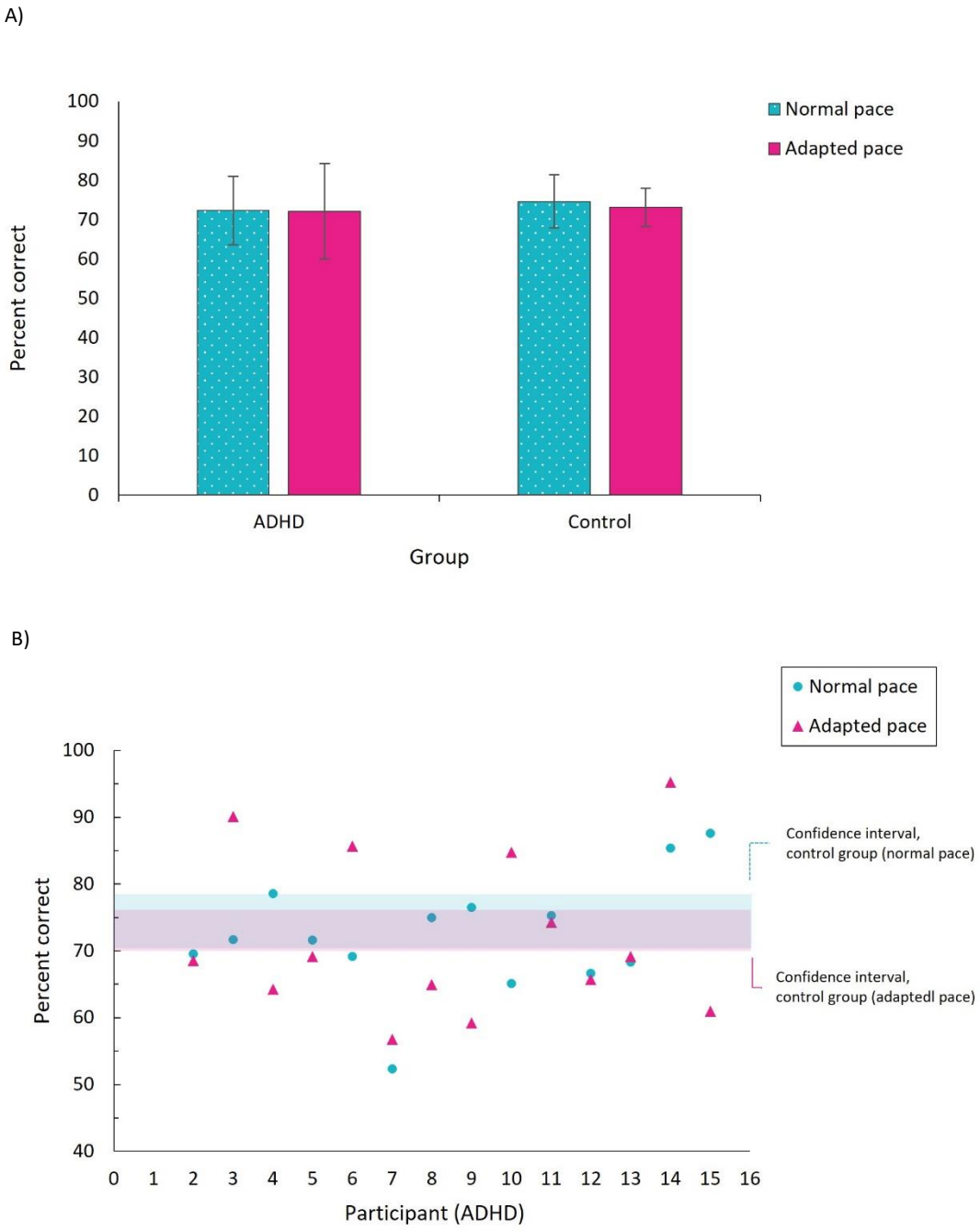

Figure 20. (A) Percentage of letters recalled in the correct positions in Experiment 3, by group and pace condition. The vertical bars represent the standard deviations. (B) Individual percentages in the ADHD group plotted against the 95% confidence intervals of the control group. The zones in light blue and pink represent the confidence intervals of the controls in the normal pace (71% - 78%) and in the adapted pace (70% - 76%), respectively.

The results of the Bayesian ANOVA showed no evidence in favour of a pace effect (BF_{10} = 0.292, error = 1.1), a group effect (BF_{10} = 0.404, error = 1.66), nor their interaction (BF_{10} = 0.042, error = 1.791), therefore the best model accounting for the data was the null. Accordingly, we found moderate evidence for the exclusion of the factors pace (BF_{excl} = 4.69) and group (BF_{excl} = 3.46) from the model, and strong evidence for the exclusion of the interaction between them (BF_{excl} = 10.86).

For the sequential Bayesian analysis, we set the alternative hypothesis as Percent $Correct_{(ADHD)}$ < Percent $Correct_{(Controls)}$ in both pace conditions. This analysis revealed fluctuations in the anecdotal range and no clear trend in the normal pace. In the adapted pace, there is a trend towards moderate evidence against the alternative hypothesis. Figure 21 shows the sequential changes in the Bayes factor.

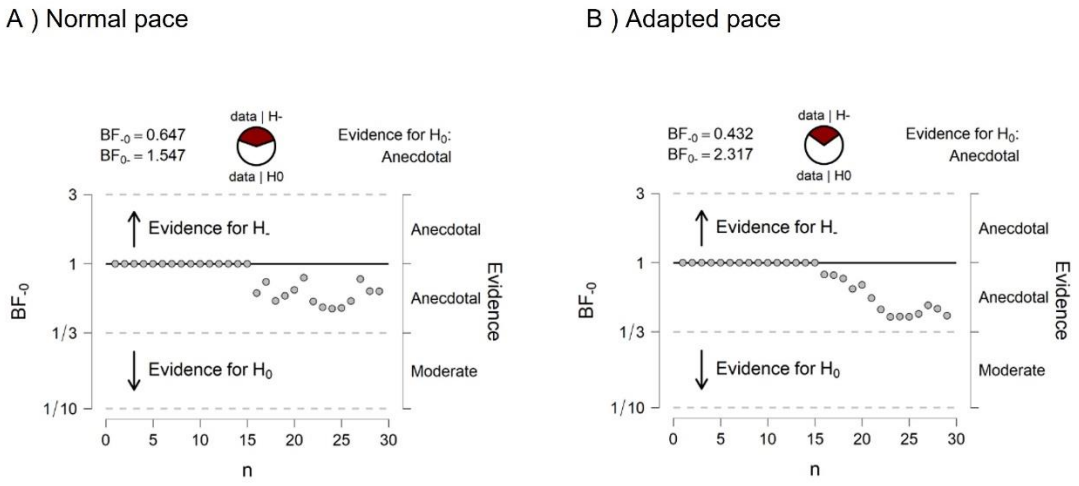

Figure 21. Sequential Bayesian analysis comparing the percentage of letters correctly recalled between groups in the normal (A) and adapted (B) pace. The alternative hypothesis was Percent $Correct_{(ADHD)}$ < Percent $Correct_{(Control)}$. In both panels A and B, the points to the right side of the figure represent the cumulative changes in the BF_{10} of the alternative hypothesis at every new data observation in the ADHD group.

Taken together, the analyses of the mean spans and the percentage of letters recalled in correct positions indicated that there were no differences in memory performance between the groups and pace conditions in Experiment 3. This result is contrary to our initial hypothesis predicting that the adapted condition would improve recall performance.

Tailoring the pace of presentation of the stimuli in the processing task to each participant's processing speed did not promote any benefits in performance for both ADHD and controls.

7.2.3. Cognitive load

According to the TBRS model of working memory, manipulating the pace of the processing component of a complex span task conducts to changes in the cognitive load (CL) that affects recall performance. Indeed, this effect was observed in Experiment 2, in which the fast pace of reading digits led to an increase of the CL and a consequent drop in performance. Because the absence of a pace effect in Experiment 3 critically contrasts with the results of Experiment 2, in which we found extreme evidence in favour of a pace effect, we ran a *post-hoc* analysis to examine whether the pace manipulation in Experiment 3 caused changes in participants' CLs. For this, we calculated the cognitive loads (CL) of participants in the two pace conditions of Experiment 3. In the normal pace, the CL was calculated by dividing the mean RT of correct responses (T_a, time of attentional grabbing) in the spatial fit processing task by 2000 ms ($T_{t(normal)}$, total time between each spatial fit, including the 500 ms of blank screen between stimuli). In the adapted pace, the CL was calculated by dividing the mean RT of correct responses in the spatial fit task (T_a) by the sum of the adapted stimuli duration and 500 ms of blank screen ($T_{t(adap)}$ = Adapted duration + 500 ms).

It is possible that the manipulation of the pace in Experiment 3 led to small variations in the CL that did not translate into real gains in memory recall. If so, we would expect to see lower CLs in the adapted pace condition for both groups of participants. Oppositely, it is also possible that the adapted pace had no impact upon the CL of participants at all. To rule out these accounts of our data, we compared the CLs across groups and pace conditions by using a Bayesian repeated-measures ANOVA.

The mean CL in the ADHD group was 0.44 (SD = 0.06) in the normal pace and 0.42 (SD = 0.07) in the adapted pace. The mean CL in the control group was 0.46 (SD = 0.03) in the normal pace and 0.39 (SD = 0.08) in the adapted pace. According to this analysis, the best model accounting for the data was the one including only the main effect of the pace (BF_{10} = 18.93, error = 0.92). The second-best model was the one including the

main effects of the pace, the group, and their interaction (BF_{10} = 16.93, error = 2.47). Because the difference between the two best models was not large, we examined the BF_{incl} of each predictor variable in the model. We found strong evidence in favour of the inclusion of the factor pace (BF_{incl} = 20.91). The inclusion of the factors group (BF_{incl} = 0.79, no evidence) and its interaction with the pace (BF_{incl} = 2.34, anecdotal evidence) was not supported by this analysis. Figure 22 shows the CLs of participants in each experimental condition.

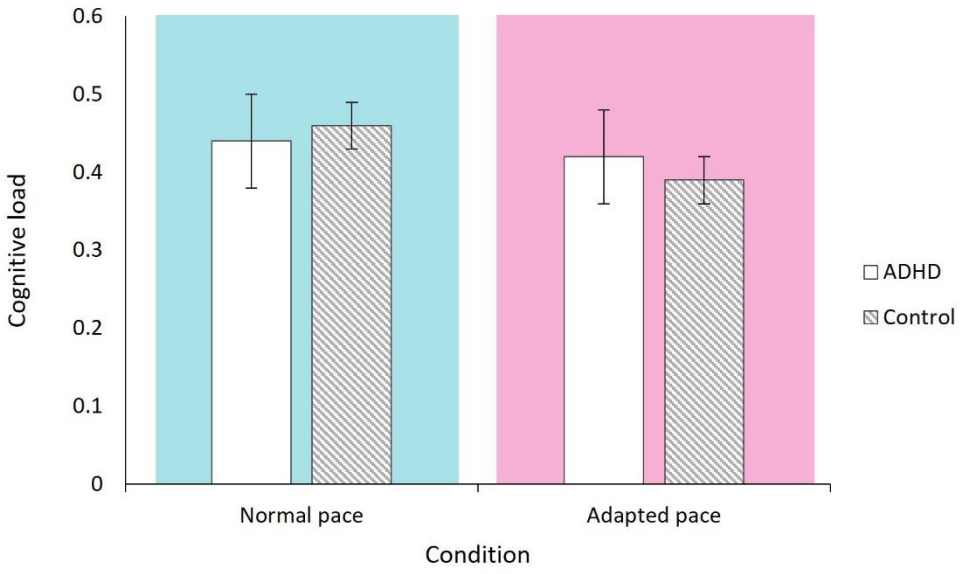

Figure 22. Mean cognitive load per group and pace condition of Experiment 3. The vertical bars represent the standard deviations.

Although the pace manipulation did not decrease the CLs dramatically (only 0.02 for ADHD and 0.07 for controls), the effect was consistent in our sample. A closer examination of the individual CLs reveals a trend of an interaction between the group and pace, as suggested by a greater number of participants in the control group that had their CL alleviated in the adapted pace. Among controls, the CL of 14 over 15 participants decreased in the adapted pace, whereas the same happened to only 8 over 14 ADHD participants. The difference in the number of ADHD and control participants whose CLs decreased in the adapted condition caused the model *pace* + *group* + *pace*group* (BF_{10}

=16.93) to be the second-best model in the Bayesian ANOVA of the CLs, not much distant from the best model including only the pace effect (BF_{10} = 18.93).

It is worth noting that the majority of the ADHD participants whose CL was alleviated in the adapted pace presented inattentive clinical symptoms (i.e., inattentive and combined subtypes). Among them, there were five with the inattentive subtype, and two with the combined subtype. Only one child in the benefited subgroup was diagnosed with the hyperactive subtype. The opposite relation between hyperactivity and increased CL in the adapted condition was also supported by the inspection of individual data points. All the remaining ADHD participants whose CL was not alleviated by the adapted pace had hyperactive-impulsive symptoms (three with the hyperactive-impulsive subtype and three with the combined subtype). For them, the adapted pace actually increased the CL of the task. Table 5 describes the relation between the clinical subtype and the changes in the CL.

Table 5

Differences in the cognitive load of ADHD participants in Experiment 3

Age	Inattention	Hyperactivity	Diagnostic subtype	$CL_{(diff)}$
13.80	80	72	Inattentive	-0.16
16.40	73	90	Inattentive	-0.09
12.75	90	45	Hyperactive	-0.08
13.80	85	89	Combined	-0.07
16.33	90	47	Combined	-0.05
13.90	57	67	Inattentive	-0.04
11.04	83	63	Inattentive	-0.02
15.96	90	81	Inattentive	-0.02
14.17	75	90	Combined	0.01
13.42	66	40	Hyperactive	0.01
14.94	44	45	Combined	0.02
11.10	53	67	Combined	0.04
12.80	65	90	Hyperactive	0.10
11.66	75	90	Hyperactive	0.11

Note. The column $CL_{(diff)}$ represents the difference between the CL in the normal pace and the CL in the adapted pace. Negative values represent a reduction of the CL in the adapted pace. The values are organized in ascending order of $CL_{(diff)}$. The columns Inattention and Hyperactivity contain the *T*-scores in the Conners-3 subscales.

The observation of the individual CLs raises the question on whether participants with higher levels of inattention benefit more from the pace manipulation, by having lighter CLs in the adapted pace than the hyperactive peers. Because inattentive participants have a slower processing speed of stimuli, it is reasonable to expect that their CLs would decrease in the adapted pace. Conversely, because hyperactive-impulsive participants have faster RTs, the consequence would be an augmentation of the CL in the adapted pace. As presented, the inspection of the individual data points permitted some speculations. In order to better account for this potential relation, we ran exploratory Bayesian correlational analyses between the CLs and the *T*-scores of inattention and hyperactivity. We included all controls and ADHD participants in the analysis. The Bayesian correlations did not reveal any evidence of a relationship between the CLs in our experimental conditions and the *T*-scores of inattention and hyperactivity in our sample (all Bayes factors smaller than 2). The correlation matrix can be found in Annex 6.

7.3. Comparison between Experiment 2 and Experiment 3

As discussed in the previous sections, the absence of a benefit of the adapted pace in Experiment 3 contradicts the finding of a strong deleterious effect of the fast pace upon memory recall in Experiment 2. The two experiments are essentially mirrored versions of the manipulation of the pace and were designed following the same rationale. The addition of a fast condition to read the digits in Experiment 2 intended to hamper performance by increasing the CL of the task. Complementarily, the addition of an adapted pace to perform the spatial fit task in Experiment 3 aimed at improving recall by decreasing the CL of the task. Moreover, the pace of presentation of stimuli in the slow condition of Experiment 2 and in the normal condition of Experiment 3 were identical, with a ratio of 0.33 between the free time (blank screen) and the duration of the stimuli of the processing task (625ms/1825ms and 500/1500ms in Experiments 2 and 3, respectively). The diverging findings between the two experiments raise the question of why recall performance was hampered by the fast pace in Experiment 2 but not improved by the adapted pace in Experiment 3.

We examined the effects of our manipulations in Experiments 2 and 3 by running Bayesian repeated-measures ANOVAs to compare the differences in the mean spans between the two levels of the pace manipulation (Slow-Fast vs. Adapted-Normal) in each group. In the ADHD group, as all participants took part in the two experiments, we set the factors Experiment (Experiment 2 vs. 3) and pace (slow/normal vs. fast/adapted) as within

factors. In the control group, because each participant took part in only one experiment, we set the Experiment (Experiment 2 vs. 3) as a between-factor and the pace (slow/normal vs. fast/adapted) as a within factor.

Both for ADHD and controls, the best model accounting for the data is the one including the main effects of the experiment, the pace, and their interaction. In the ADHD group, the Bayes factor of the full model was $BF_{10} = 2.24903 \times 10^5$ (error = 2.07) and we found decisive evidence for the inclusion of the factors experiment ($BF_{incl} = 8.7524 \times 10^4$), pace ($BF_{incl} = 214.99$), and their interaction ($BF_{incl} = 581.182$) in the model. In the control group, the Bayes factor of the full model was $BF_{10} = 17.482 \times 10^4$ (error = 2.84) and we found decisive evidence for the inclusion of the factors experiment ($BF_{incl} = 244.76$), pace ($BF_{incl} = 2.315 \times 10^3$), and their interaction ($BF_{incl} = 255.91$) in the model. It is worth noting that the strongest evidence was for the inclusion of the experiment among ADHD participants ($BF_{incl} = 8.7524 \times 10^4$), whereas, among controls, the strongest evidence was in favour of the pace ($BF_{incl} = 2.315 \times 10^3$). Coupled with the observation of the plots (Figure 20), this difference in the BF_{incl} between ADHD and controls suggests that the pace effect in ADHD participants was more variable depending on the experiment. The fast pace in Experiment 2 clearly impaired the recall performance of both ADHD and controls, but while controls (Figure 23, B) were unaffected by the adapted pace in Experiment 3, ADHD showed a trend to an improvement in performance (Figure 23, A).

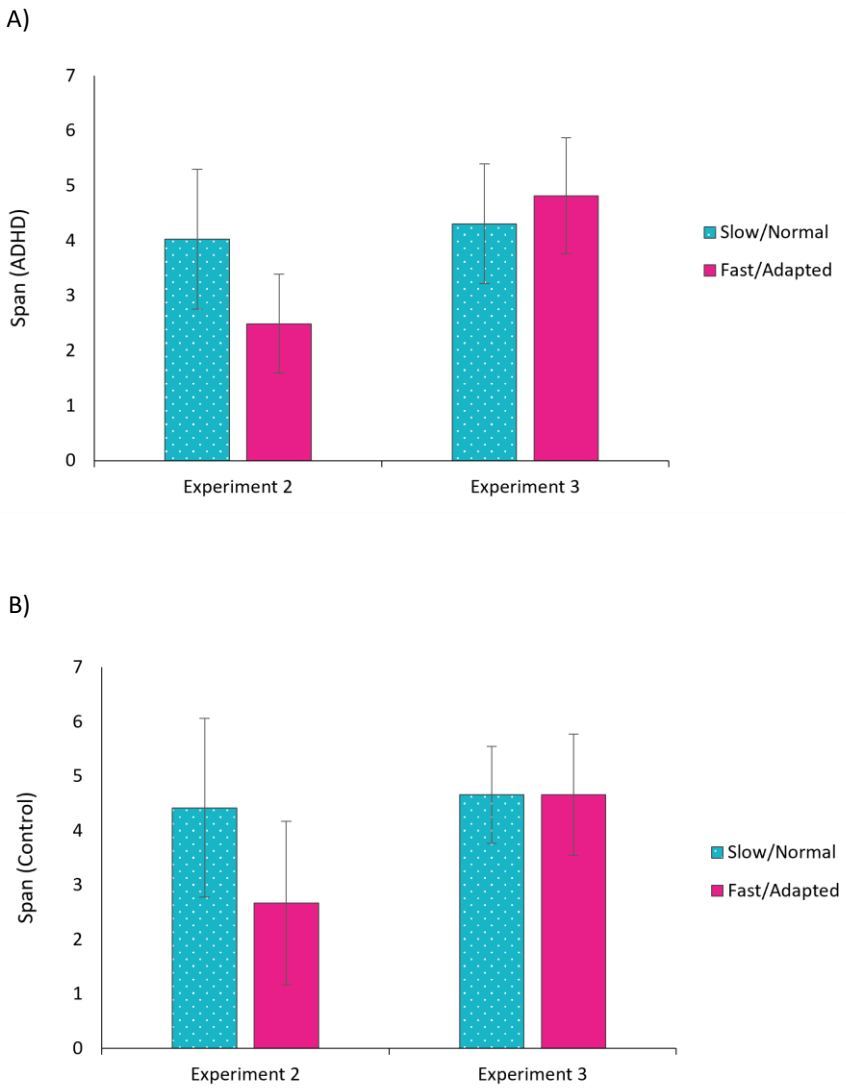

Figure 23. Comparison between the effect of the pace manipulation in experiment 2 and 3 in the ADHD (A) and control (B) groups. The two levels of the pace manipulation in each experiment are collapsed: slow pace (Experiment 2) is collapsed with normal pace (Experiment 3) and fast pace (Experiment 3) with adapted pace (Experiment 3). The vertical lines represent the standard deviations.

7.4. Discussion

The results of Experiment 3 showed that there was no difference in recall performance in the normal and adapted pace for both groups of participants, contrary to our hypothesis. Despite the absence of a pace effect upon the dependent variables, we found strong evidence that the adapted pace caused the CL to decrease, as we had predicted. In other words, tailoring the pace of presentation of the stimuli in the processing task made the CL lighter for participants, but it did not promote benefits in recall performance. These results sharply contrast with the ones of Experiment 2, in which an

increase of the CL caused by the fast pace was followed by a decrease in memory performance.

The comparison between Experiment 2 and Experiment 3 confirmed that the effects of the manipulation of the pace was different between the two tasks, and that this difference was modulated by the group. Although we did not find a group effect both in Experiment 2 and Experiment 3, a trend for a better performance of the controls in the normal condition of Experiment 2. Moreover, fewer ADHD benefiting from the adapted pace in Experiment 3. These might have contributed for the inclusion of the factor group and the interaction group x experiment in the model.

An important question is why the pace manipulation did not affect recall performance in Experiment 3. If ADHD participants are prone to the CL effect as their typically developing peers (as seen in Experiment 2), why would this effect not be detected in an optimal condition in which they performed the task at their own rhythm? Specifically concerning ADHD participants, a straightforward explanation that the effect of the adapted pace was very heterogeneous, with some participants greatly benefitting from it, whereas other were unaffected or even disrupted. The examination of the individual data points (Figure 20) illustrates this argument. Another possible explanation is that their performance was already very close to controls in the normal pace, which suggests that they were performing at ceiling and that they did not need the adapted pace to improve their performance.

Indeed, the observation of participants' mean RT in the spatial fit task showed that ADHD participants were not slower than controls in any of the experimental conditions, not even in the baseline RT measured during the training phase. We compared the mean RTs between groups during the training phase using a Bayesian *T*-tests to rule out this explanation. We found no evidence that ADHD had a slower baseline RT to respond to the spatial fit task (BF_{10} = 0.616, error = 6.47 x 10^{-5}). In terms of absolute values, ADHD participants were slightly *faster* than controls during the training, with a mean RT of 1350 ms (SD = 500 ms) versus a mean RT of 1548 ms (SD = 899 ms) in controls. Also, the large dispersion of the individual baseline RT in the control group is somehow rather unexpected. Through pure observation, we noticed that some participants in the control group tented to seek the experimenter's approval before responding to the spatial fit during the training (e.g., by asking "can I click now?" or answering aloud to the experimenter before touching the mouse), whereas all ADHD – thanks to their diminished inhibition – promptly started the task as soon as the mouse was given to them. The results of our

Experiment 3 call for replication and encourage further investigations, possibly with some modifications in tasks' implementation and instructions.

To summarize our results, the manipulation of the pace in Experiment 3 alleviated the CL in the adapted pace – a finding that mirrors the results of Experiment 2. Nevertheless, this reduction of the CL was not followed by an increase in memory recall as we hypothesized. The absence of a pace effect in Experiment 3 contradicts Experiment 2. Finally, congruent to findings of Experiment 1 and Experiment 2, we did not find evidence of a group difference between ADHD and controls upon working memory performance. At least in our sample, ADHD participants were as able as their typically developing peers in recognition memory for colours and verbal serial recall.

Chapter 8

General discussion

The interplay between attention and working memory is one of the most important topics in cognitive psychology and we proposed an experimental approach to investigate it in children with ADHD according to the predictions of the TBRS model. Specifically, we conducted three experiments to test if this population could orient attention to perceptual stimuli and to mental representations held in working memory (Experiment 1) and if they could use attentional refreshing to maintain items in working memory (Experiments 2 and 3). Our central hypothesis was that, given their persistent inattentive symptoms, children with ADHD would have difficulties orienting attention retrospectively and refreshing items in working memory compared to typically developing controls, which could account for their working memory deficits.

The idea of modelling cognitive processes in clinical populations is not novel (e.g., Weigard et al., 2018; see the volume edited by Neufeld, 2007, for a global perspective), however, testing these populations to advance theoretical models in Cognitive Science is rather rare in the recent literature. Few attempts to study models of working memory through experimentation in clinical populations were made in the past decade. To our best knowledge, only two studies specifically targeted the ADHD population for that purpose. One is the work by Ortega et al. (2020), who tested a sample of children with ADHD to investigate whether binding operations in the episodic buffer do not require additional attentional resources, as proposed by the multimodal component model of working memory (Allen, Badelley, & Hitch, 2006; Allen, Hitch, & Badelley 2009; Karlsen et al., 2010). In the study of Ortega et al. (2006), participants performed a visuospatial change detection task with a single feature (only shape) and a binding condition (colour-shape). Because no differences in performance between ADHD and controls were observed in the binding condition, the authors suggest that visuospatial binding in the episodic buffer is not an attentional demanding operation. The second is the work by Weigard and Huang-Pollock (2017) mentioned in Chapters 6 and 7. These authors investigated the role of processing speed upon working memory performance based on the predictions of the

TBRS model by testing children with ADHD in a visuospatial complex span task. Results showed that slower processing speeds hampered performance in both groups and no interactions group x speed conditions were observed. The authors suggest that slower processing speed account for working memory deficits in ADHD and provided evidence that children with ADHD are subject to the cognitive load effect predicted by the TBRS in a visuospatial working memory task.

The common reasoning between the studies of Ortega et al. (2020) and Weigard and Huang-Pollock is to test a population acknowledged for having attentional deficits and slowed processing speed to investigate specific assumptions about the functioning of working memory. The present thesis joins their studies in the effort to examine inner mechanisms of working memory via experimentation in the population of children with ADHD. The benefits of such a research agenda are bi-directional. First, it helps the community of clinical researchers to better locate the purported dysfunctions of working memory in ADHD, by providing mechanistic explanations based on basic psychological processes that can be assessed, quantified, and manipulated via experimental tasks. Second, it allows the community of basic researchers in working memory to verify whether theoretical assumptions and phenomena observed in typically developing individuals generalize to this population.

As presented in Chapters 5, 6, and 7, the results of Experiments 1 and 2 did not confirm our hypotheses, and the results of Experiment 3 were inconclusive. We interpreted this set of results as preliminary evidence that attentional orienting (Experiment 1) and attentional refreshing (Experiment 2) were not the sources of ADHD-related working memory deficits in our sample. Next, we will discuss the impact of these results upon the theoretical understanding of working memory and the clinical understanding of ADHD.

8.1. Attention-based maintenance in working memory

As extensively explained in Chapter 1, different theoretical models acknowledge the existence of two maintenance mechanisms in working memory: phonological (or articulatory) rehearsal and attention-based maintenance. We will restrict our discussion to attention-based maintenance, as it is the theme of this doctoral thesis.

The three models addressed in Chapter 1 agree that attention acts as a bottleneck to working memory performance and that it can be used to maintain information in this cognitive system. Nevertheless, the models diverge on the precise nature of attention-based

maintenance and the targets of its operation. For the TBRS model (Barrouillet et al., 2004; Barrouillet & Camos, 2015) and the embedded-processes model (Cowan, 1988, 1999), attention can be used to maintain information in any sensorial modality and representational code, whereas the multicomponent model considers that attention is mainly used to maintain multimodal information in the episodic buffer (Baddeley et al., 2011).

There are also differences in how attention is conceptualized by the authors of such models. The TBRS and the multicomponent models tend to consider attention as an energetic-like resource of the system, in a sense that the operations of the system cannot take place if not "fuelled" by it. According to this account, attention is either considered as a shared resource between processing and maintenance requirements (TBRS model) or as a source of the executive control carried out by a homuncular central executive (multicomponent model). The Embedded-processes model, in its turn, sees attention as a selective focus that determines the relevance of information by selecting a subset of items from long-term knowledge during the execution of a given task. The view of attention as a selective focus is more akin to metaphors of attention as a zoom lens or a spotlight than to an energetic-like resource.

These two conceptions of attention (resource vs. selective focus) influence how authors conceive the effects of its limitations in working memory. For Cowan, the operations in working memory are restricted by the size of the attentional focus, that is, by how many items attention it can select and shuttle inside and outside the focus (Cowan et al., 2021, p. 45). This limitation can be understood as a quantitative one, in the sense that the focus of attention is essentially limited in the number of mental representations it can simultaneously contain. The authors of the TBRS model, in their turn, consider that working memory is limited by the time available to switch attention between processing and maintaining information. Because attention can only be used to perform one operation at a time, the amount of free time between operations will be a constricting factor. The availability of time to switch attention from processing to maintenance determines the efficiency of attentional refreshing – conceptualized as the rapid and sequential switching of attention between these operations. This limitation can be understood as a temporal one, that is, working memory is essentially limited by the time course of attentional demands during a cognitive task (Barrouillet & Camos, 2015; 2021).

Among these three theoretical views, the TBRS model proposes an operational definition of cognitive load that makes the translation from predictions to experimental testing easy. Also, the explicit relation between the cognitive load and recall performance in complex span tasks, mediated by the efficiency of attentional refreshing, allows us to make conclusions about the use of this strategy by participants. One main question guided the directions of this thesis: Are clinical populations subjected to the same constraints to the use of attentional refreshing in working memory than typically developing individuals? The observation of a cognitive load effect in Experiment 2, together with results reported by Weigard and Huang-Pollock (2017), suggest that yes. Moreover, these results suggest that refreshing can be used both in visuospatial (Weigard & Huang-Pollock, 2017) and verbal tasks (our Experiment 2), as observed in typically developing populations. In this regard, the results of Experiment 2 are aligned with the views of the TBRS and the embedded-processes models that attention-based maintenance is a supra-modal strategy in working memory, acting upon verbal and visuospatial content.

The cognitive load effect was not observed in Experiment 3, which was designed to provide supplementary evidence to Experiment 2. While discussing the results of our Experiment 3 and facing the absence of a group effect, we suggested that our participants probably relied more heavily on articulatory rehearsal than participants in the study of Weigard and Huang-Pollock (2017), given the different nature of the two tasks – theirs was a visuospatial span, whereas ours was a letter span. The parallel use of phonological rehearsal and attentional refreshing in our adapted reading span task could account for the differences between the two studies regarding a group effect. This interpretation is in line with the proposal of the TBRS that verbal information is maintained in working memory via both attentional refreshing and articulatory rehearsal (Camos, 2015, 2017).

Our results are accommodated in the proposal of the TBRS model, but they can also be understood under the light of the other two theories presented in this thesis. First, the mean spans of ADHD and control groups in Experiment 2 and 3 coincide with the limits of the focus of attention, estimated between 3-4 slots of information (Cowan et al., 2005) and highlighted by the embedded-processes model. According to this theoretical framework, the absence of a group difference in the mean spans suggests that ADHD performance was not limited by an inferior number of slots to which information can be allotted – in other words, *how many* items can be consciously represented at once in the attentional focus.

Second, one could argue that serial recall of letters involves the maintenance of bound episodic representations (which letter is in which serial position) in an episodic buffer. As we mentioned before, the authors of the multicomponent model defend that attention is not required to maintain bound information in the buffer (Allen, Badelley, & Hitch, 2006; Allen, Hitch, & Badelley 2009; Karlsen et al., 2010), and the study of Ortega et al. (2020) showed that, indeed, ADHD participants had the same memory performance for visuospatial-bound material (colour-shape bindings in spatial locations) than typically developing children. According to this line of reasoning, the absence of a group effect in Experiments 2 and 3 could be explained exclusively due to the intrinsic properties of the episodic buffer, i.e., by the fact that it does not rely on attentional resources to maintain letters bound to serial positions. In this framework, any purported lack of attention affecting working memory functioning would not be detected by such task and a cross-modal serial recall task would be more adequate.

Our experiments were not designed to test the predictions of the three models of working memory presented in this thesis (Chapter 1), therefore comparisons between them are out of the reach of our data (for model comparisons, see Doherty et al., 2019). The effort to interpret our results according to different theoretical frameworks is a reasoning exercise serving to refrain hasty conclusions and to deepen our understanding of two other influent accounts of working memory in the literature, the embedded-processes, and the multicomponent models. We hope that such exercise instigates other researchers to study models of working memory via experimental testing in clinical populations, as done in the present doctoral thesis.

8.2. Working memory as an explanation of ADHD

The working memory model of ADHD, proposed by Rapport et al. (Rapport, Chung, et al., 2001; Rapport, Kofler et al., 2008; Alderson et al., 2010), acknowledges the importance of working memory deficits in ADHD and defends that temporal decay is the cause of behavioural symptoms of inattention and hyperactivity. Rapport's model is mainly concerned with explaining behavioural symptoms of inattention and hyperactivity as an effect of working memory deficits, not the working memory deficits *per se.* According to their model, rapid decay in working memory causes behavioural disinhibition (the source of hyperactive-impulsive symptoms) because a chain of processes relying on working memory operations cannot take place. Such operations are short-term

maintenance (e.g., remembering an instruction), processing the immediate context (thus responding accordingly to the environment), and retrieving information from long-term memory (e.g., recalling internalized rules). As for the inattentive symptoms, the authors propose that constantly diverting attention to stimuli (external or internal) counteracts rapid decay and keeps the input rate of information constant in working memory. Stimulation seeking in the form of high levels of motor activity also counteracts decay and compensate for the low input rates in working memory.

Approximations between Rapport's model and the theories of working memory presented in this thesis can be made. The more evident one is that Rapport posits rapid decay as the cause of behavioural symptoms of ADHD, therefore assuming the existence of a deficit in maintenance mechanisms of working memory – despite not explicitly mentioning them in the model. This deficit could affect articulatory rehearsal and/or attentional refreshing, which are two maintenance strategies unanimously acknowledged by the multimodal, the embedded-processes, and the TBRS model of working memory. Previous works (Bolden et al., 2012; Kofler et al., 2010) already showed that children with ADHD have more difficulties in using phonological rehearsal, so we designed our Experiments 2 and 3 to test if an impaired use of attentional refreshing accounts for difficulties in working memory in ADHD.

Another approximation concerns the conceptualization of attention in Rapport's model and its similarities to the concept of a focus of attention in Cowan's embedded-processes model of working memory. Rapport conceptualizes attention as a targeting mechanism of the contents of working memory at a given moment, which is similar to Cowan's proposal of an attentional focus holding information in a heightened state of activation and available to conscious inspection. Because it is not possible not to think about anything, Rapport considers that inattentive symptoms in ADHD are the behavioural manifestation of a rapid change of the contents of working memory – or, in the embedded processes' vocabulary, the transient activation of information in the focus of attention.

It is a matter of theoretical alignment if we look at the problem as one of the transient representational states of information in working memory or one of implementation of maintenance strategies. The former approach would ask questions such as "For how long activated representations linger in the focus of attention in ADHD?" or "How many representations can ADHD children hold in the focus of attention?". The second approach would ask questions such as "How effective is the use of attentional

refreshing and/or phonological rehearsal?", as we did in this thesis. Naturally, one could argue that the implementation of maintenance strategies in working memory is a means of conserving mental representations activated in a conscious attentional focus, thus that the debate is simply a matter of lexical choice. We do not partake of this simplistic view. Addressing the issue of rapid attentional decay in ADHD under the assumptions the embedded-processes model would require different experimental manipulations than the ones in this thesis.

Finally, this thesis did not address the theoretical models of ADHD in the literature directly. Rather, we used them to guide our reasoning throughout this doctoral research; Rapport's working memory of ADHD especially influenced our reasoning. As a by-product of this influence, we believe that our experiments can offer insights on how to study Rapport's model of ADHD via experimentation, particularly on how some inner mechanisms of working memory can lead to a rapid decay in this clinical population. Next, we will present the conclusions driven about the two working memory mechanisms under investigation in this thesis: attentional orienting and attentional refreshing.

8.3. The locus of working memory deficits in ADHD

8.3.1. Cue-based attentional orienting in ADHD

Our first approach to locate working memory deficits in ADHD was to examine two stages of information processing: encoding and maintenance. We did it by using two cueing paradigms that bias attentional orienting during these processing stages, i.e., pre-cues and retro-cues, retrospectively. Attention acts as a bottleneck to working memory performance not only for the maintenance of information therein and the executive control of the system. It also acts as a gateway to the sensorial information entering the system. Attending to given locations or visual features in the perceptual space enhances memory retrieval of information encoded at these locations (Makovski & Jiang, 2007; Schmidt et al., 2002; Theeuwes et al., 2011). Similarly, orienting attention to a spatial location within mental representations also enhances working memory performance, a phenomenon called the retro-cue effect (Griffin & Nobre, 2003; Makovski & Jiang, 2015; van Moorselaar et al., 2015; see Souza et al., 2013 for a review).

Attentional orienting modulates maintenance in working memory but it is not a maintenance mechanism in itself. The decision to start our investigation by examining cue-

based orienting effects in ADHD was based on two basic principles. First, cue-based orienting is a simpler mechanism than attentional refreshing: it has been observed in macaques (Bowman et al., 1993) and it develops rapidly during the first year of human babies (Ross-Sheehy et al., 2015). Second, the combined use of pre-cues and retro-cues in the same task offered a well-delimited starting point to our attempt to find the locus of a working memory deficit in ADHD, that is, during encoding and/or maintenance.

Our hypothesis for Experiment 1 was that both groups would benefit from pre-cues but only typically developing participants would benefit from retro-cues. We observed cueing benefits in both groups in all experimental conditions, and there were no differences in performance between ADHD participants and controls. Indeed, studies from the 1990s showed that attentional orienting to perceptual stimuli is not impaired in ADHD (Huang-pollock et al., 2003; Jonkman et al., 2005), and a meta-analytical review by Huang-Polock et al. (2005) showed that ADHD is not characterized by an impairment of visuospatial orienting. The presence of a pre-cueing benefit in the ADHD group is consistent with these reports in the literature. Concerning the observed retro-cue benefits in the ADHD group, we are not aware of studies in the literature that specifically examined this phenomenon. To our best knowledge, this thesis provides the first evidence that children with ADHD are prone to the retro-cue effect, which indicates that they can voluntarily orient attention to mental representations to boost working memory performance.

Our interpretation of the results in Experiment 1 is that cue-based attentional orienting is intact in children with ADHD, suggesting that two important properties of attention, selection and modulation, are preserved in this population. Selection refers to how attention selects targets to be encoded to, maintained in, or retrieved from working memory. Modulation refers to the property of influencing what happens to the selected targets, i.e., how well or how fast the target information is processed, how fast and accurate the response is given, and whether the information will be remembered in the future (Chun et al., 2011). The concomitant observation of pre- and retro-cue benefits in the working memory of children with ADHD strengthens the evidence of functional similarities between external and internal attention (Chun & Johnson, 2011). In other words, our results allowed the simultaneous observation of some intrinsic properties of external and internal attention in a population acknowledged by an attentional deficit.

8.3.2. Attentional refreshing in ADHD

Experiments 2 and 3 were designed specifically to tackle the use of attentional refreshing to maintain information in working memory. As explained, the efficiency of attentional refreshing depends on the time available to switch attention from processing demands to maintenance demands in working memory. If attention is free from processing demands, it will be used to refresh items maintained in working memory and counteract temporal decay. The ratio between the time of the attentional capture by a processing operation and the total time to perform it is called the cognitive load. The TBRS model has specific predictions on how the cognitive load affects the use of attentional refreshing, thus impacting working memory performance. Under high cognitive load conditions, the time to refresh items is reduced thus performance drops. Experiments 2 and 3, therefore, varied the pace of presentation of stimuli in the processing task to manipulate the cognitive load, therefore affecting the efficiency of attentional refreshing.

Experiment 2 was based on the reading digit span task proposed by Barrouillet et al. (2009). Participants were required to memorize increasingly longer sequences of letters and to read digits between each letter in the sequence. We manipulated the pace of presentation of the digits so that the cognitive load was low in one condition (slow pace) and high in the other (fast pace). According to our reasoning, if children with ADHD do not use attentional refreshing to maintain items in working memory, then their performance should not be affected by an increase in the cognitive load at the fast pace. The results showed that, contrary to our prediction, there was a strong effect of the pace manipulation upon performance in both groups of participants, an absence of a group effect, and no interaction between pace and group. These results indicate that memory performance was similar in both groups and, more importantly, that the fast pace impaired performance in ADHD participants to the same extent as controls.

The deleterious effect of the fast pace upon recall in the ADHD group suggest that the working memory of children and adolescents with ADHD are subject to the same time constraints as typically developing children and adults. As explained, the cognitive load effect indexes one's ability to use free time to use attention to refresh items and protect memory representations from decay. Its presence in the ADHD group suggests that this population can perform attentional refreshing to maintain verbal material in working memory. Moreover, it is consistent with a previous observation of a cognitive load effect

in a visuospatial complex span task (Weigard & Huang-Pollock) and the theoretical position that attention-based maintenance in working memory is used for verbal and visuospatial material, defended by the TBRS and the embedded-processes models. We highlight the fact that we did not instruct participants to use attentional refreshing, therefore the cognitive load effect in Experiment 2 show the spontaneous use of this strategy by children with ADHD.

The results discussed so far showed that an increase of the cognitive load impaired memory performance in children with ADHD. Experiment 3 was designed to test the complementary prediction that a reduction of the cognitive load would cause an increase in memory performance. To do so, we replaced the digits by a spatial fit task in which participants were required to answer via mouse clicks if a horizontal bar fit the gap between two squares. The implementation of a mouse response allowed us to calculate each participant's baseline RT in the spatial fit task and use it to tailor the presentation of the stimuli to their own processing speed. The adaptation of the pace mitigates individual differences in processing speed and equalizes the cognitive load between participants. Thus, Experiment 3 had two pace conditions: a normal pace, in which the cognitive load was variable between participants, and an adapted pace, in which the cognitive load was equal to all participants.

Experiments 2 and 3 were designed in parallel, thus our initial hypothesis of a group difference between ADHD and controls had not yet been contradicted by the results of Experiment 2. Our hypothesis for Experiment 3 was that the adapted condition would benefit ADHD participants and abolish group differences in performances. The results of Experiment 3 were inconclusive, as we found neither a group effect nor a pace effect. The absence of a group effect is consistent with the results of Experiments 1 and 2, but the absence of a pace effect upon performance conflicts with the results of Experiment 2. Although the adapted pace did not yield benefits in performance in Experiment 3, it caused a slight, but significant decrease in the cognitive load of participants, as we had predicted. Nevertheless, this modest reduction in the cognitive load was not translated into gains in the mean span and percentage of correct letters recalled. Some factors might have contributed to the absence of a cognitive load effect in performance in Experiment 3. First, the baseline RTs in the spatial fit task was not different between the two groups, causing our manipulation of the pace to be ineffective. Second, ADHD and controls had the same cognitive load at the normal pace, suggesting that ADHD participants did not need an

adaptation of the pace to improve performance. We will discuss more the absence of group differences in section 8.4.

To end, the set of results in this thesis suggest that ADHD-related deficits in working memory are not attributable to an inability to use attention retrospectively (Experiment 1) or attentional refreshing (Experiment 2). Rather, we showed evidence that these processes were intact in our sample of children with ADHD. Next, we will address another potential source of the working memory difficulties in ADHD: a developmental delay in the acquisition and optimization of attentional orienting and attentional refreshing.

8.3.3. A developmental delay in ADHD?

We already explained in Chapter 1 that attentional refreshing starts to be used by typically developing children around age 7 and is optimized until adolescence (Barrouillet et al., 2009; Camos & Barrouillet, 2011; Gaillard et al., 2011). The same trend for the benefit in using retro-cues to boost working memory performance was reported by Shimi et al. (2014), albeit evidence of a growth of the retro-cue benefit during childhood is scarcer in the literature. We did not test different age groups to assess the development of these abilities in ADHD in the present thesis, but we believe that our results can indirectly inform us about a developmental trend.

The absence of a group effect in our Experiments 1 (the colour-cued recognition) and 2 (the reading-digit span task) suggest that our sample of ADHD participants were equally capable as controls of the same age to benefit from retrospective attentional orienting (Experiment 1, retro-cue condition) and more time to use attentional refreshing (Experiment 2, slow pace condition). It seems that our ADHD participants were not developmentally delayed compared to the control group in the acquisition of these two abilities. The results of the Bayesian ANOVA considering the age of the participants as a covariate strengthens this interpretation. In sum, our results provide indirect evidence that attentional refreshing and retrospective attentional orienting in ADHD follow the same developmental trend as in typically developing children. Future studies should compare different age groups of ADHD children to ascertain this hypothesis.

To summarize this section of the General Discussion, this thesis showed evidence that: 1) children and adolescents with ADHD can use retro-cues to boost performance during a recognition task (Experiment 1) and are prone to the cognitive load effect in a complex span task predicted by the TBRS model (Experiment 2); 2) they are able to use attentional refreshing to maintain verbal material (Experiment 2); 3) they do not seem to

be delayed in the acquisition of such abilities compared to typically developing children (Experiments 1 and 2).

8.4. Current limitations and future perspectives

One limitation of this study is the large variability in the age and symptoms of our participants. We chose an age group from 10 to 16 years because there is evidence in the literature that cue-based attentional orienting and attentional refreshing are mechanisms fully developed in typically developing children from the beginning of adolescence (Shimi et al., 2014; Barrouillet et al., 2009). The choice of such a large age range was not purely based on evidence in the literature, though; it had also a component of necessity face to difficulties in recruiting participants in the ADHD group. The same applies to the inclusion of participants diagnosed with all subtypes of ADHD in our sample. Ideally, we targeted only participants diagnosed with the inattentive and combined subtype, but it was very difficult to reach a minimum sample size with this inclusion criteria. We believe that these two factors introduced greater variability in our data and probably accounted for the absence of the expected group differences between ADHD and control groups in all three experiments.

The use of a verbal complex span task can also have contributed to the absence of a group effect in Experiments 2 and 3, although it was a purposeful methodological choice. As previously mentioned, Weigard and Huang-Pollock (2017) found a group effect in a visuospatial complex span task with a similar manipulation than ours (i.e., varying the processing speed of participants to affect the cognitive load). We explained before that visuospatial working memory tasks are more sensitive to the presence of an ADHD diagnosis than verbal tasks (Chapter 2) and that this evidence is supported by meta-analytical studies (Martinussen et al., 2005; Kasper et al., 2012; Willcutt et al., 2005). We do not believe that our results surmount all the evidence in the literature showing an ADHD-related deficit in working memory. Rather, our Experiments 2 and 3 could not be sensitive enough to detect group differences between ADHD and controls because they required only memory for verbal content. The set of stimuli in the reading digit span task (Experiment 2) entirely consisted in verbal material (i.e., letters and digits). The adapted reading span task (Experiment 3) also required participants to recall verbal content (letters), despite its visuospatial processing component (make spatial size judgments). A possible way to rule out this explanation is to combine our experimental design with one of Weigard

and Huang-Pollock (2017), for instance, by introducing our spatial fit processing task in a spatial span task. The cognitive load could be manipulated either by varying the pace of presentation of spatial fit stimuli (our approach) or by making the judgements in the spatial fit task more difficult to slow down participant's processing speed (Weigard and Huang-Pollock's approach).

The explanation for the absence of a group effect in Experiments 2 and 3 due to the use of verbal material raises the question of why the effect did not appear in Experiment 1 either. Experiment 1 was a colour recognition task combined with location cues; therefore, it tapped the visuospatial domain of working memory. A possible explanation is that Experiment 1 involved a discrete colour recognition, while ADHD-related difficulties might reflect impairments in the precision of working memory representations. Recognition tasks detect differences in performance in a dichotomous way: the probe is either recognized or not by the participant, implying an all-nothing retrieval process. If ADHD participants have a precision deficit instead, the high discriminability of the colours we used (red, pink, green, orange, violet, blue instead of, e.g., crimson, magenta, carmine, and vermilion, all of which belong to the category "red") made the task too easy to detect their working memory deficit. This is a speculative suggestion; we are not aware of studies that specifically tested if working memory representations in ADHD are less precise than typically developing individuals. To do so, future studies should use delayed estimation tasks, like the colour-wheel, to investigate if the retro-cue effect replicates in children with ADHD.

Still on the absence of a group effect in Experiment 1, it is also possible that maintaining static arrays of one-feature visual information does not pose difficulties to children with ADHD. Spatial attention is fundamental to bind single features in the visual field into object-based representations, and it has been compared to a "glue" that binds visual information together (Treisman, 1988, 1998; Treisman & Gelade, 1980). Therefore, any attentional deficit in working memory would be more easily detected by a task requiring the correct identification of more than one visual feature at the same spatial location. For example, a change detection task with locations bound to colours and shapes, like the one carried out by Ortega et al. (2020), is suitable for this purpose. A recent study by Alderson et al. (2021) used a recognition task for only colours, shapes, and colour-shape binding and found consistent group differences between ADHD and controls (mean age of 9.8 years) in all conditions.

For the reasons explained above, we advocate prudence in interpreting the absence of a group effect in our results. They should not be taken as evidence that children with ADHD perform working memory tasks equally well as their typically developing peers. This rapid conclusion is contradicted by many results in the literature (Martinussen et al., 2005; Kasper et al., 2012; Willcutt et al., 2005), including the study by Weigard and Huang-Polock (2017) who used the same experimental paradigm as we did in Experiments 2 and 3. Instead, our results should be interpreted as preliminary evidence that ADHD-related difficulties in working memory do not stem from dysfunctions of attentional orienting nor attentional refreshing, as assessed by structured experimental tasks in a controlled environment.

Our results cannot rule out the possibility that a more pervasive attentional deficit interfering in other processes (e.g., decision making, response selection, vigilance, awareness) affects working memory performance in more ecological settings. Performing in real-life contexts is much more challenging and depends on many other psychological processes such as intrinsic and extrinsic motivation, reacting to reinforcement and punishment, inhibiting responses, etc. – all these basic psychological phenomena are altered in ADHD, as discussed in Chapter 2. It is impossible to control all these variables in experimental settings and it is very difficult to design an experiment tackling more than one of them at a time, let alone their interaction. We advise clinicians to seek a global comprehension of the functioning of other basic other psychological processes in ADHD in order to interpret the studies about the working memory functioning in this population, including the ones presented in this thesis and those using the neuropsychological testing approach.

Conclusion

This thesis was an attempt to provide a mechanistic explanation to the attentional deficit in working memory of children with ADHD by translating the predictions of the TBRS model of working memory to this clinical population. In terms of the inner processes of working memory, the experiments presented in this thesis suggest that, at least in our sample, the ADHD-related deficits in working memory are not located in internal attentional orienting nor in the use of attentional refreshing. The clinical impact is that our results offer a clue to where the attentional deficit lie in the working memory of children with ADHD – we showed evidence suggesting where it is *not*. In terms of theoretical impacts, our results showed preliminary evidence that the retro-cue effect and the cognitive load effect replicate in a sample of children and adolescents with ADHD, suggesting that this population is able to orient attention and use attentional refreshing to boost maintenance in working memory. As always in science, initial evidence must be interpreted with caution. Future studies should aim to replicate our results in bigger samples with more control over the age and diagnostic variability of participants. We encourage researchers to use classical experimental paradigms to locate the working memory deficits in ADHD, for the experimental method is the only one able to provide the refined mechanistic explanations that are dear to cognitive psychology. "Where to look next?" to find the locus of working memory difficulties in ADHD is an open-ended question that we look forward to seeing answered. We expect that it can bring insightful knowledge on the functioning of working memory along the way to solve it.

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Annex 1

Complete sample characterization

Table 6

Complete sample characterization of the study.

	Sex	Age	ADHD subtype	Previously diagnosed	IN	HY	LP	EF	AG	PR	Global Index	AN	AH	CD	OD	Probability score	Medication (psychostimulant)
ADHD Group	F	11.1	Combined	Yes	90	90	72	83	63	53	83	90	90	58	67	0.99	No
	F	15.4	Hyperactive	Yes	73	90	63	54	56	43	78	67	90	45	52	0.97	Yes
	F	12.0	Inattentive	Yes	90	45	76	72	59	53	65	86	45	58	55	0.71	Yes
	F	15.9	Inattentive	Yes	90	47	90	76	44	43	49	85	47	45	43	0.87	No
	M	12.8	Combined	Yes	80	72	72	70	83	42	71	77	71	72	77	0.99	Yes
	M	13.2	Combined	Yes	85	89	77	84	61	42	90	87	89	48	69	0.99	Yes
	M	13.5	Hyperactive	Yes	57	67	55	45	51	84	57	51	69	45	51	0.41	Yes
	M	10.6	Inattentive	Yes	83	63	79	75	50	64	77	78	61	43	56	0.87	No
	M	15.6	Combined	Yes	90	81	86	82	59	80	90	90	90	47	74	0.99	Yes
	M	11.4	Combined	Yes	75	90	47	56	90	74	90	77	90	90	90	0.99	Yes
	M	13.8	Combined	Yes	75	90	65	65	69	64	84	72	90	66	69	0.99	Yes
	M	13.1	Inattentive	Yes	66	40	70	47	41	69	42	52	40	43	40	0.51	Yes
	M	14.6	Inattentive	Yes	44	45	42	40	50	41	51	43	43	47	61	0.11	Yes
	M	11.0	Hyperactive	Yes	53	67	47	40	43	42	74	44	62	49	49	0.56	Yes
	M	12.6	Hyperactive	Yes	65	90	50	51	67	81	83	62	86	61	61	0.91	No
Control group (No symptoms)	F	15.9	-	No	52	58	48	45	44	59	51	50	59	45	45	0.51	-
	F	14.3	-	No	40	51	41	49	48	48	56	45	50	50	48	0.11	-
	F	12.8	-	No	45	42	50	45	47	43	45	41	43	51	42	0.11	-
	F	12.6	-	No	40	42	40	40	43	43	40	40	41	43	42	0.11	-
	F	13.0	-	No	44	48	45	42	44	49	52	41	43	52	44	0.11	-
	F	14.9	-	No	52	48	50	48	61	48	55	49	46	55	55	0.11	-
	F	12.7	-	No	49	49	55	45	43	58	43	45	55	51	41	0.11	-
	F	13.4	-	No	41	44	54	40	48	58	41	41	43	52	47	0.11	-
	F	12.0	-	No	49	60	40	42	47	43	56	47	55	43	47	0.29	-

	Sex	Age	ADHD subtype	Previously diagnosed	IN	HY	LP	EF	AG	PR	Global Index	AN	AH	CD	OD	Probability score	Medication (psychostimulant)
Control group (No symptoms)	F	11.5	-	No	40	43	44	40	42	53	40	40	45	44	41	0.11	-
	F	10.4	-	No	40	44	40	40	43	42	43	40	45	44	40	0.11	-
	F	10.9	-	No	51	55	42	49	56	48	58	47	53	52	59	0.11	-
	F	10.3	-	No	43	53	40	40	64	42	50	40	47	60	52	0.11	-
	F	10.9	-	No	45	44	40	57	43	42	45	44	40	52	40	0.11	-
	F	11.5	-	No	48	58	51	44	42	43	43	52	61	44	46	0.11	-
	F	12.0	-	No	40	51	42	40	42	43	45	47	53	44	41	0.29	-
	F	12.1	-	No	55	47	52	49	54	53	52	50	46	43	52	0.11	-
	M	12.5	-	No	46	53	43	45	52	90	57	55	52	50	66	0.51	-
	M	14.5	-	No	42	45	40	42	41	47	51	41	43	50	40	0.11	-
	M	15.0	-	No	47	41	53	44	51	47	46	53	41	51	53	0.11	-
	M	12.4	-	No	44	56	43	42	58	75	52	50	56	50	66	0.11	-
	M	15.4	-	No	40	41	40	40	43	43	40	40	47	43	41	0.11	-
	M	15.6	-	No	45	41	45	57	45	43	46	57	41	43	47	0.41	-
	M	12.1	-	No	42	44	41	47	43	59	42	42	43	44	44	0.11	-
	M	11.9	-	No	44	40	40	40	43	55	40	41	40	43	40	0.11	-
	M	11.1	-	No	44	45	42	47	52	55	53	48	41	49	65	0.11	-
	M	10.6	-	No	55	60	52	43	51	43	57	57	64	49	51	0.41	-
	M	11.2	-	No	53	57	56	40	43	49	45	40	64	43	40	0.11	-
	M	12.0	-	No	46	55	54	45	49	42	45	48	57	42	42	0.11	-
	M	12.3	-	No	53	58	48	48	55	53	57	50	58	50	58	0.11	-
	M	11.6	-	No	63	59	60	52	55	68	51	58	67	42	49	0.41	-
	F	11.2	-	No	73	64	63	69	55	53	68	65	69	51	67	0.64	-
	F	12.9	-	No	49	62	47	45	68	73	53	54	64	51	57	0.41	-
	F	12.9	-	No	53	67	50	51	47	43	53	56	67	43	44	0.56	-
	F	14.6	-	No	64	58	50	58	54	75	60	63	48	55	60	0.64	-
	F	15.5	-	No	54	71	53	49	59	48	65	50	67	50	56	0.29	-

	Sex	Age	ADHD subtype	Previously diagnosed	IN	HY	LP	EF	AG	PR	Global Index	AN	AH	CD	OD	Probability score	
Control (Inconclusive)	F	15.0	-	No	56	62	66	54	61	59	63	53	62	50	57	0.11	-
	F	12.4	-	No	53	62	50	53	47	43	53	56	64	43	49	0.29	-
	F	12.0	-	No	58	67	68	53	61	48	71	52	64	51	57	0.29	-
	F	10.6	-	No	76	44	69	54	64	42	55	63	42	44	40	0.71	-
	F	12.6	-	No	68	45	52	49	47	73	71	66	43	51	52	0.51	-
	F	11.1	-	No	63	58	40	58	63	63	75	47	53	88	78	0.51	-
	F	11.4	-	No	53	66	53	48	80	53	63	49	64	80	70	0.11	-
	F	11.4	-	No	60	60	47	55	42	43	53	63	59	44	52	0.41	-
	M	15.6	-	No	45	68	50	40	45	43	60	41	69	47	47	0.41	-
	M	12.1	-	No	69	56	45	51	86	42	76	58	50	72	74	0.64	-
	M	10.9	-	No	59	72	57	41	48	49	71	47	73	49	51	0.56	-
	M	11.7	-	No	61	55	47	54	43	90	56	56	57	43	43	0.64	-
	M	11.9	-	No	63	53	60	63	52	62	61	54	54	49	49	0.29	-
Excluded participats																	Exclusion criterion(a)
	F	15.8	-	No	69	66	58	58	59	43	72	70	73	50	60	0.71	Suspicion of ADHD
	F	15.0	-	No	65	90	55	58	71	43	83	56	90	59	64	0.64	Suspicion of ADHD
	F	14.8	-	No	73	52	56	58	47	80	53	63	50	45	52	0.71	Suspicion of ADHD
	F	10.7	-	No	63	80	74	54	56	42	68	72	74	52	61	0.77	Suspicion of ADHD
	F	10.3	-	No	59	90	42	46	64	59	78	54	90	52	67	0.82	Suspicion of ADHD
	F	10.2	-	No	76	90	69	70	43	48	76	70	90	44	52	0.91	Suspicion of ADHD
	F	12.5	-	No	47	47	45	40	61	43	53	47	43	51	67	0.29	Incomplete task
	F	9.87	-	No	-	-	-	-	-	-	-	-	-	-	-	-	Age
	F	9.81	-	No	-	-	-	-	-	-	-	-	-	-	-	-	Age
	F	11.1	-	No	60	58	53	51	63	43	70	52	56	51	67	0.11	Incomplete task
	M	13.5	-	No	69	75	42	56	41	42	58	65	75	48	48	0.94	Suspicion of ADHD

	Sex	Age	ADHD subtype	Previously diagnosed	IN	HY	LP	EF	AG	PR	GI	AN	AH	CD	OD	Probability score	
	M	13.5	-	No	60	66	45	62	58	53	63	63	73	52	59	0.91	Suspicion of ADHD
	M	15.7	-	No	69	79	42	40	48	47	63	57	90	43	47	0.64	Suspicion of ADHD
	M	11.2	-	No	71	90	79	65	49	55	72	77	90	43	52	0.99	Suspicion of ADHD
	M	10.3	-	No	79	58	69	65	51	67	60	89	60	54	55	0.91	Suspicion of ADHD
	M	15.0	-	No	79	50	88	72	54	85	55	82	60	43	53	0.77	Suspicion of ADHD
	M	14.9	-	Yes	44	44	40	41	41	41	42	43	43	43	40	0.11	Previous diagnosis
	M	1.03	-	No	-	-	-	-	-	-	-	-	-	-	-	-	Age
	M	10.6	-	No	69	68	64	69	82	84	68	68	69	71	75	0.94	Suspicion of ADHD

Note. The table contains data of all participants tested in the study, including those who were excluded. Participants are grouped according to the group and the presence/absence of symptoms detected in the Conners-3 Parent subscale. The ages are represented in years. The subgroup "Controls(Inconclusive)" correspond to control participants whose parents gave inconsistent responses in the symptoms scales. The columns from IN, HY, LP, EF, AG, PR, GI, AN, AH, CD, and OD contain the *T*-scores in the respective Conners-3 Parent subscales and the clinical threshold is a *T*-score of 61, corresponding to the 85th percentile. IN = Inattention; HY = Hyperactivity/Impulsivity; LP = Learning Problems; EF = Executive Functioning; AG = Defiance/Aggression; PR = Peer Relations; GI = Global Index; AN = DSM-IV-TR ADHD Inattentive; AH = DSM-IV-TR ADHD Hyperactive-Impulsive; CD = Conduct Disorder; OD = Oppositional Defiant Disorder.

Table 7

Sample characterization in Experiment 1

	Sex	Age (years)	ADHD subtype	Inattention	*T*-Score Hyperactive/ Impulsivity	*T*-Score Probability Index
ADHD group	F	11.1	Combined	90	90	0.99
	F	15.4	Hyperactive	73	90	0.97
	F	12.0	Inattentive	90	45	0.71
	F	15.9	Inattentive	90	47	0.87
	M	12.8	Combined	80	72	0.99
	M	13.2	Combined	85	89	0.99
	M	13.5	Hyperactive	57	67	0.41
	M	10.6	Inattentive	83	63	0.87
	M	15.6	Combined	90	81	0.99
	M	11.4	Combined	75	90	0.99
	M	13.8	Combined	75	90	0.99
	M	13.1	Inattentive	66	40	0.51
	M	14.6	Inattentive	44	45	0.11
	M	11.0	Hyperactive	53	67	0.56
	M	12.6	Hyperactive	65	90	0.91
Subtotals / Means	15 (4 female)	13.1 (1.7)		74.4 (14.7)	71.96 (19.3)	0.79 (0.27)
Control group	**F**	**15.9**		**52**	**58**	**0.51**
	F	14.3		40	51	0.11
	F	12.8		45	42	0.11
	F	13.0		44	48	0.11
	F	14.9		52	48	0.11
	F	13.4		41	44	0.11
	F	**10.4**		**40**	**44**	**0.11**
	F	11.2		73	64	0.64
	F	12.9		53	67	0.56
	F	15.0		56	62	0.11
	F	12.4		53	62	0.29
	F	11.4		53	66	0.11
	F	11.4		60	60	0.41
	M	14.5		42	45	0.11
	M	12.4		44	56	0.11
	M	11.9		44	40	0.11
	M	10.6		55	60	0.41
	M	15.6		45	68	0.41
	M	**12.1**		**69**	**56**	**0.64**
Subtotals / Means	19 (13 females)	13.0 (1.6)		50.6 (9.4)	54.8(9.2)	0.26(0.2)

Note. Only the variables of interest from the Conners-3 Parent are included in the table. Values between parentheses correspond to standard deviations. Lines in bold represent outliers not included in the analyses.

Table 8

Sample characterization in Experiment 2

	Sex	Age (years)	ADHD subtype	Inattention	*T*-score Hyperactive/ Impulsivity	*T*-score Probability Index
ADHD group	F	11.2	Combined	90	90	0.99
	F	15.5	Hyperactive	73	90	0.97
	F	12.2	Inattentive	90	45	0.71
	F	16.0	Inattentive	90	47	0.87
	M	12.9	Combined	80	72	0.99
	M	13.3	Combined	85	89	0.99
	M	13.5	Hyperactive	57	67	0.41
	M	1.7	Inattentive	83	63	0.87
	M	15.7	Combined	90	81	0.99
	M	11.6	Combined	75	90	0.99
	M	13.9	Combined	75	90	0.99
	M	13.1	Inattentive	66	40	0.51
	M	14.6	Inattentive	44	45	0.11
	M	11.9	Hyperactive	53	67	0.56
	M	12.7	Hyperactive	65	90	0.91
Subtotals/ Means	15 (4 female)	13.2 (1.7)		74.4 (14.7)	71.96 (19.3)	0.79 (0.27)
Control group	F	16.0	-	52	58	0.51
	F	14.4	-	40	51	0.11
	F	12.8	-	45	42	0.11
	F	12.7	-	40	42	0.11
	F	12.8	-	49	49	0.11
	F	10.4	-	43	53	0.11
	F	10.9	-	45	44	0.11
	F	12.0	-	40	51	0.29
	F	11.2	-	73	64	0.64
	F	14.6	-	64	58	0.64
	F	15.6	-	54	71	0.29
	F	10.7	-	76	44	0.71
	F	12.6	-	68	45	0.51
	M	12.5	-	46	53	0.51
	M	15.0	-	47	41	0.11
	M	15.4	-	40	41	0.11
	M	**15.7**	**-**	**45**	**68**	**0.41**
	M	10.9	-	59	72	0.56
	M	11.8	-	61	55	0.64
Subtotals/ Means	19 (13 females)	13.1 (1.9)		51.9 (11.6)	52.7 (10.2)	0.3 (0.23)

Note. Only the variables of interest from the Conners-3 Parent are included in the table. Values between parentheses correspond to standard deviations. Lines in bold represent outliers not included in the analyses.

Table 9

Sample characterization in Experiment 3

	Sex	Age (years)	ADHD subtype	Inattention	*T*-score Hyperactive/ Impulsivity	*T*-score Probability Index
ADHD group	F	16.4	Hyperactive	73	90	0.97
	F	12.8	Inattentive	90	45	0.71
	F	16.3	Inattentive	90	47	0.87
	M	13.8	Combined	80	72	0.99
	M	13.8	Combined	85	89	0.99
	M	13.9	Hyperactive	57	67	0.41
	M	11.0	Inattentive	83	63	0.87
	M	16.0	Combined	90	81	0.99
	M	11.7	Combined	75	90	0.99
	M	14.2	Combined	75	90	0.99
	M	13.4	Inattentive	66	40	0.51
	M	14.9	Inattentive	44	45	0.11
	M	11.1	Hyperactive	53	67	0.56
	M	12.8	Hyperactive	65	90	0.91
Subtotals/ Means	14 (3 females)	13.7 (1.7)		73.3 (14.6)	69.7(19.3)	0.8(0.27)
Control group	F	16.5		52	58	0.51
	F	12.8		49	60	0.29
	F	11.5		40	43	0.11
	F	10.9		51	55	0.11
	F	11.6		48	58	0.11
	F	12.1		55	47	0.11
	F	13.0		49	62	0.41
	F	12.7		58	67	0.29
	F	**11.1**		**63**	**58**	**0.51**
	M	15.6		45	41	0.41
	M	12.1		42	44	0.11
	M	11.1		44	45	0.11
	M	11.2		53	57	0.11
	M	12.7		46	55	0.11
	M	12.3		53	58	0.11
	M	11.9		63	53	0.29
	M	11.7		63	59	0.41
Subtotals/ Means	17 (9 females)	12.4 (1.5)		51.4 (7.2)	54.1 (7.45)	0.2 (0.15)

Note. Only the variables of interest from the Conners-3 Parent are included in the table. Values between parentheses correspond to standard deviations. Lines in bold represent outliers not included in the analyses.

Annex 2

Consent forms

UNIVERSITÉ DE FRIBOURG
UNIVERSITÄT FREIBURG

ATTENTION AND WORKING MEMORY IN CHILDREN WITH ATTENTIONAL AND HYPERACTIVITY DISORDER

« La mémoire et l'attention dans le TDAH » (titre usuel abrégé)

Ce projet de recherche est organisé par : Prof. Valérie Camos, Chair du Développement Cognitive, Département de Psychologie, Université de Fribourg

Madame, Monsieur,

Par la présente, nous souhaiterions vous informer de notre projet de recherche. Votre enfant étant encore mineur, il ne peut donner son consentement pour le projet prévu. Par conséquent, nous vous faisons parvenir la présente feuille d'information, qui vous permettra de vérifier si vous pouvez accepter une participation de sa part. En effet, vous pouvez accorder votre consentement en qualité de parents/ représentants légaux.

Information détaillée

1. Objectif du projet

Dans ce projet, nous cherchons à mieux comprendre le fonctionnement cognitif des enfants avec un trouble du déficit de l'attention avec ou sans hyperactivité (TDAH), spécifiquement sur ses habiletés de mémoire de travail et d'attention en comparaison aux enfants ayant un développement typique.

2. Sélection des personnes pouvant participer

La participation est ouverte à toutes les personnes qui souffrent d'un trouble du déficit de l'attention avec ou sans hyperactivité (TDAH) ayant entre 10 et 16 ans, et qui ont été diagnostiqués par un spécialiste. Par ailleurs, les participants ne peuvent pas présenter un de ces troubles suivants : trouble intellectuel, trouble psychotique, trouble du langage, démence ou tout autre trouble qui empêcherait de suivre la procédure de l'étude. Les participants peuvent avoir une médication comportant un psychostimulant prescrit pour le TDAH, mais pas d'autres psychotropes.

3. Informations générales sur le projet

La mémoire de travail est l'habileté cognitive de retenir et manipuler des informations à court terme (de quelques secondes à quelques minutes). Elle est absolument essentielle pour accomplir des tâches comme les calculs mathématiques, la compréhension verbale ou le raisonnement. Soit à l'école (lire, écrire, faire des calculs) ou dans la vie quotidienne (se rappeler d'une liste de courses, le parcours à la maison, ranger ses affaires), la mémoire de travail est impliquée quasiment dans toutes nos activités. Récemment, la recherche scientifique a suggéré que les enfants portant un TDAH auraient de différentes capacités de mémoire de travail, qui conduiraient à leurs difficultés attentionnelles et de contrôle du comportement. Nous cherchons à comprendre quels sont les composants de la mémoire de travail qui seraient différents chez les enfants et adolescents présentant un TDAH par rapport aux enfants sans TDAH.

Ce projet demande la participation à des expériences sur ordinateur. Les tâches ressemblent à un jeu de mémoire, où l'enfant doit mémoriser des stimuli (par exemple, des couleurs, ou

des lettres). Les tâches seront ludiques et adaptées à la capacité des enfants et des adolescents.

La durée totale de notre projet est de trois ans (2019-2022), durée pendant laquelle nous mènerons plusieurs expériences et qui inclut toutes les phases de la recherche (de la récolte des données, aux analyses des données et des résultats, à l'écriture de la thèse de doctorat). Vous avez le choix de faire participer votre enfant à une ou plusieurs expériences de notre projet (décrites en détail ci-dessous). Vous n'avez pas à décider maintenant du nombre d'expériences auquel votre enfant
participera. Vous pourrez faire ce choix plus tard, par exemple, après la participation à une expérience ou même nous recontacter dans le futur.

Le nombre approximatif de participants de ce projet est soixante personnes (trente participants portant un TDAH et trente participants de développement typique). Ce projet a une portée nationale et est réalisé dans le respect de la législation suisse. La commission d'éthique compétente (CER-VD, Commission cantonale d'éthique de la recherche sur l'être humain) a contrôlé et autorisé le projet.

4. Déroulement

Cette étude comprend une première phase de sélection de participants et une seconde phase de participation à trois tâches de mémoire. En tant que parents, vous recevrez par courrier postal cinque questionnaires qui évaluent le fonctionnement de votre enfant dans différents domaines (par exemple : fonctions exécutives, émotionnel, comportemental, aptitudes sociales). Ces questionnaires prennent environ 40 minutes pour être remplis. Ils servent à la vérification des critères d'inclusion à l'étude pour chaque participant, et ne fournissent pas un diagnostic clinique. Après avoir rempli les questionnaires, nous vous demanderons de nous les renvoyer par voie postale à l'aide de l'une enveloppe timbrée que nous vous avons fournie. Après le retour des questionnaires, une première prise de contact avec l'investigatrice de l'étude sera effectuée par téléphone et/ou émail, afin de voir si cette étude est réalisable ou non pour vous et votre enfant (temps consacré, explications, motivation, contre-indications, prise de rendez-vous, etc.). Si vous êtes d'accord de participer à l'étude et si votre enfant remplit les critères d'inclusion, l'investigatrice de l'étude vous contactera pour fixer les dates des rendez-vous et préciser les aspects pratiques de la participation de votre enfant.

Cette recherche comprend trois types de tâches de mémoire : un pour étudier la mémoire de travail visuo-spatiale (exemple : les formats, les couleurs, les emplacements); un pour étudier la mémoire de travail verbal (exemple : les mots, les syllabes, les lettres); et un troisième afin de vérifier si les participants peuvent apprendre à utiliser des stratégies pour améliorer leurs performances sur les tâches de mémoire, en fonction des résultats à obtenir dans les deux expériences précédentes. La durée moyenne de chaque expérience n'excède pas trente minutes, et chaque expérience sera menée dans une session individuelle dans un lieu de votre choix (maison de la famille, école, etc.). Le lieu de réalisation des expériences devra être un lieu silencieux où l'enfant se sentira confortable et calme.

Expérience de mémoire visuo-spatiale : le participant s'assied à 60 cm de distance de l'écran d'ordinateur. Chaque essai comporte les événements suivants : quatre objets de différentes couleurs sont rapidement affichés à l'écran. Le participant doit mémoriser ces objets. Après un court intervalle, un seul objet est affiché au centre de l'écran et le participant doit répondre si cet objet figurait parmi les quatre vus précédemment en appuyant sur une touche du clavier. Dans certains cas, une flèche pointera un emplacement de l'écran et le participant devra faire bien attention à l'objet placé à cet emplacement. L'expérimentateur restera avec le participant pendant toute la session expérimentale.

Expérience de mémoire verbale : le participant s'assied à 60 cm de distance de l'écran de l'ordinateur. A chaque essai, il devra mémoriser des séries de 2 à 8 consonnes. Chaque

consonne sera suivie d'une tâche de jugement où le participant devra juger si un chiffre est pair ou impair. Plusieurs chiffres seront présentés les uns après les autres soit à un rythme rapide (1000 ms par chiffre), soit à un rythme lent (3000 ms par chiffre), afin de faire varier la demande attentionnelle pendant la tâche. De plus, la tâche de jugement des chiffres sera exécutée soit silencieusement en appuyant sur des touches du clavier, soit en appuyant sur ces touches mais en répétant les syllabes «ba bi bou» de façon continue tout au long de l'expérience. Cette dernière condition a pour but de bloquer les stratégies verbales et encourager l'usage de l'attention pendant la tâche de mémoire.

Expérience sur les stratégies mémorielles : Au vu des résultats des deux précédentes expériences, nous choisirons de reprendre soit la tâche de mémoire visuo-spatiale soit la tache de mémoire verbale afin d'étudier les stratégies mémorielles. La tâche sera la même que celle précédemment décrite, mais nous expliquerons aux participants la stratégie à utiliser pendant les instructions.

5. Bénéfices pour les participants

La participation à cette étude n'apportera pas de bénéfice personnel au participant. Cependant, sa participation permettra de faire avancer la recherche et de mieux comprendre ce qui sous-tend les difficultés mémorielles des enfants et adolescents ayant un TDAH. Les résultats des recherches pourraient être importants pour les personnes touchées par le même trouble.

6. Droits

La participation au projet est facultative. Si votre enfant ne souhaite pas participer ou si vous, en tant que parent/représentant légal, revenez ultérieurement sur votre décision à ce sujet, vous n'aurez pas à vous justifier. La participation peut être retirée à tout moment. Vous pouvez à tout moment poser vos questions concernant la participation et le projet. A cette fin, veuillez-vous adresser à la personne indiquée à la fin de la présente feuille d'information.

7. Obligations

Si votre enfant participe à l'étude, il faut observer certaines règles pour son bon déroulement :

- De suivre les instructions et de remplir les exigences de l'investigateur lors de la participation aux expériences proposées, spécifiquement :
    - rester assis(e) devant l'ordinateur lors de l'exécution de la tâche ;
    - observer des images, lettres, chiffres, couleurs sur l'écran de l'ordinateur et les mémoriser ;
    - prononcer à voix haute les syllabes « ba bi bou » quand sollicité ;
    - répondre aux tâches en appuyant sur les touches du clavier ou en utilisant la souris.

- Le cas échéant, ne pas prendre de médication comportant un psychostimulant 24 heures avant la participation dans l'étude.

8. Risques

Les participants ne seront exposés à aucun risque d'intégrité physique ou psychologique au cours de la recherche. Nous estimons que cette recherche représente une charge minime pour nos participants, étant donné que les expériences sont courtes (moins de 30 minutes) et qu'elles consistent en des tâches de mémoire assistées par ordinateur qui n'impliquent aucune procédure invasive, douloureuse ou ne demandent pas trop d'efforts cognitifs. Par contre, si votre enfant est sous traitement médical, nous vous demanderons de faire cesser la prise du médicament 24h avant les expériences. Si le fait d'arrêter le traitement présente un risque

pour votre enfant, l'accord du médecin traitant sera demandé et ce-dernier vous informera des conséquences liées à l'arrêt de prise de ritaline.

9. Résultats

Le but de cette recherche n'est pas de se substituer à une investigation médicale afin de déterminer la présence ou non d'un diagnostic comme un trouble de l'attention avec/sans hyperactivité et impulsivité (TDAH). Les questionnaires utilisés dans cette étude fournissent des informations utiles sur le plan scientifique, mais ceci sans poser de diagnostics cliniques. La procédure diagnostique est réalisée dans le cadre d'une investigation médicale complète incluant le recueil d'informations et l'observation clinique lors de consultations, les questionnaires apportant des informations complémentaires.

La responsable du projet vous communiquera en cas de découvertes fortuites de difficultés importantes de votre enfant qui pourraient peut-être évoquer la piste d'un autre trouble correspondant à des catégories diagnostiques connues ou en conflit avec son diagnostic préalable de TDAH. Nous vous proposons de nous préciser si vous souhaitez que le médecin traitant de votre enfant ou le médecin référent de l'étude, le Dr Michel Bader, soit informé afin de vous proposer des compléments d'investigation. La responsable du projet vous avisera, en votre qualité de parent et de représentant légal du participant, de toutes nouvelles découvertes susceptibles d'influer sur la sécurité de votre enfant et, ainsi, sur votre consentement à participer.

Les résultats de cette recherche seront traités en groupe, c'est-à-dire que aucun rapport individuel sur la performance de votre enfant dans les expériences vous sera fourni après l'étude. Par contre, nous vous fournirons un rapport général par écrit des résultats et conclusions de la recherche sur le fonctionnement cognitif des enfants portant un TDAH.

10. Traitement confidentiel des données et des échantillons

Pour ce projet, nous enregistrerons les données personnelles (âge, sexe), médicales (présence ou non d'un diagnostic de TDAH, prise ou non de médication), les résultats des questionnaires, et les réponses du participant (taux de réponses correctes et temps de réponse) dans les expériences proposées. Seulement la Prof. Camos, responsable de l'étude, et Mme. Superbia Guimarães, l'investigatrice, peuvent consulter ces données sous une forme non codée, et ce exclusivement pour pouvoir accomplir les tâches nécessaires au déroulement du projet. Ces informations sont codées au moment du relevé. Le codage signifie que toutes les données permettant d'identifier les participants sont effacées et remplacées par un code. Il n'est pas possible de relier les données au participant sans le code, qui reste en permanence au sein du Département de Psychologie de l'Université de Fribourg. Parfois, les journaux scientifiques exigent la transmission de données individuelles (données brutes). Si des données individuelles doivent être transmises, elles sont toujours codées avec un numéro et ne permettent donc pas d'identifier le participant.

Au cas où une découverte fortuite se produit lors de l'analyse des données (voir point 9 au-dessus), si vous le souhaitez et l'autorisez, nous retransmettrons le résultat des échelles au Docteur Michel Bader ou au médecin traitant de votre enfant. Toutes les personnes impliquées dans le projet sont tenues au secret professionnel.

Durant son déroulement, le projet peut faire l'objet d'inspections par la commission d'éthique compétente ou par l'institution qui a commandé le projet. Pour les besoins de ces inspections, le responsable du projet doit alors retransmettre les données personnelles et médicales.

11. Retrait du projet

Le participant peut à tout moment se retirer de l'étude s'il le souhaite ou si vous en décidiez ainsi en tant que parent / représentant(e) légal. Si le participant décide de se retirer au cours de la passation d'une expérience, les données de cette expérience seront effacées au moment

même de l'arrêt. Si vous nous le demandez ultérieurement, les données recueillies pourront être effacées avant la fin de l'étude.

Pendant le projet, nous conserverons une liste contenant le nom des participants, leurs contacts et le code d'identification des fichiers de données. À la fin du projet, la liste des participants avec le code reliant ses données ainsi que leurs contacts sera détruite, mais nous conserverons les fichiers contenant leurs temps de réponse et ses réponses dans les expériences à l'Université de Fribourg pendant dix ans, mais personne ne pourra retrouver à qui se rapportent ces données.

12. Rémunération des participants
Si vous participez à ce projet, vous ne recevrez pour cela aucune rémunération.

13. Responsabilité civile
Les dommages liés au projet de recherche sont couverts par l'Université de Fribourg. Les conditions et la procédure sont réglées par la loi.

14. Financement
Le projet est financé par le Département de Psychologie de l'Université de Fribourg et le Programme de Bourses d'Excellence de la Confédération Suisse (Commission Fédérale des Bourses pour les Étudiants étrangers) dans le cadre d'un doctorat mené à l'Université de Fribourg.

15. Interlocuteurs
En cas de doutes, de craintes ou d'urgences pendant ou après le projet, vous pouvez vous adresser à tout moment à l'un des interlocuteurs suivants :

Prof. Valérie Camos, directrice de l'étude:
Département de Psychologie, Université de Fribourg,
Rue P.-A.-de Faucigny, 2, 1700, Fribourg, bureau C. 3-115
Téléphone : (41) 026 300 7675
Email : valerie.camos@unifr.ch

Luísa Superbia Guimarães, investigatrice :
Département de Psychologie, Université de Fribourg,
Rue P.-A.-de Faucigny, 2, 1700, Fribourg, bureau C. 3-113
Téléphone : (41) 026 300 76 82
Portable : (41) 076 816 86 72
Email : luisa.superguimaraes@unifr.ch

DÉCLARATION DE CONSENTEMENT

Déclaration écrite de consentement pour la participation à un projet de recherche
Veuillez lire attentivement ce formulaire. N'hésitez pas à poser des questions lorsque vous ne comprenez pas ou que vous souhaitez obtenir des précisions. Pour la participation de votre enfant, votre consentement écrit est nécessaire.

Numéro BASEC du projet de recherche (après soumission à la commission d'éthique compétente) :	2019-02325
Titre (scientifique et usuel) :	Attention and working memory in children with attentional and hyperactivity disorder "La mémoire et l'attention dans le TDAH »
Institution responsable (responsable du projet et adresse complète) :	Université de Fribourg, Département de Psychologie, Rue P.-A.-de Faucigny, 2, 1700 Fribourg
Lieu de réalisation :	Fribourg, Rue P.-A.-de Faucigny, 2, 1700 Fribourg
Responsable du projet sur place : Nom et prénom en capitales d'imprimerie :	Prof. Dr. Valérie Camos Directrice de l'étude Université de Fribourg
Participant : Nom et prénom en capitales d'imprimerie : Date de naissance :	☐ fille ☐ garçon

- En ma qualité de parent ou représentant(e) légal du participant susmentionné, j'ai obtenu les informations écrites et orales de la part de l'investigateur sur les objectifs, le déroulement du projet ainsi que les avantages, inconvénients et risques possibles.
- Je confirme prendre la décision pour le participant susmentionné qu'il participe au projet de recherche. En son nom, j'accepte les informations écrites et orales. Par ailleurs, j'ai eu assez de temps pour prendre cette décision.
- Je confirme qu'un entretien d'information entre le participant susmentionné et l'investigateur a eu lieu, et que les procédures de cette étude ont été expliquées.
- J'ai reçu les réponses aux questions que j'ai posées en relation avec la participation à ce projet, je conserve la feuille d'information datée du 16.05.2020, version 5, et reçois une copie de ma déclaration de consentement.
- J'accepte que les spécialistes compétents dirigeant ce projet, et ceux de la commission d'éthique compétente puissent consulter les données non codées des participants afin de procéder à des contrôles, à condition toutefois que la confidentialité de ces données soit strictement assurée.
- Je sais que les données personnelles ne peuvent être transmises que sous une forme anonyme et à des fins de recherche dans le cadre de ce projet.

- Au nom du participant, je peux, à tout moment et sans avoir à me justifier, révoquer mon consentement à la participation, sans que cette décision n'ait de répercussions défavorables sur lui. Les données seront détruites si vous vous retirez avant la fin de l'étude. Ensuite, les données seront anonymisées (ré-identification impossible) et conservées.
- La responsabilité civile de l'Université de Fribourg couvre les dommages pouvant découler du projet.
- Je suis conscient(e) que les obligations mentionnées dans la feuille d'information doivent être remplies pendant toute la durée du projet.

- Je souhaite que le médecin traitant de mon enfant ou que le médecin référent de l'étude, le Docteur Michel Bader, soit informé pour me proposer des compléments d'investigation.

Le médecin traitant de mon enfant	OUI ( )	NON ( )
Le Docteur Michel Bader	OUI ( )	NON ( )

Le cas échéant, veuillez nous communiquer les informations de contact de votre médecin traitant :

_____

Veuillez s'il vous plaît nous communiquer vos informations de contact :

Adresse (avec NPA) :

Numéro de téléphone :

E-mail :

Lieu, date	Nom et prénom en capitales d'imprimerie Relation avec le participant (père ou mère, etc.) : Signature du proche/du représentant légal/des parents :

Attestation de l'investigateur : Par la présente, j'atteste avoir expliqué au proche/au représentant légal/aux parents du participant la nature, l'importance et la portée du projet.

Je déclare satisfaire à toutes les obligations légales en relation avec ce projet. Si je devais, à n'importe quelque moment du projet, prendre connaissance d'éléments susceptibles d'influer sur le consentement du participant à prendre part au projet, je m'engage à informer immédiatement son proche/son représentant légal/ses parents.

Lieu, date	Luísa Superbia Guimarães (Investigatrice assurant l'information) Signature de l'investigatrice

UNIVERSITÉ DE FRIBOURG
UNIVERSITÄT FREIBURG

PARTICIPATION À L'ÉTUDE : L'ATTENTION ET LA MÉMOIRE DANS LE TDAH

Que va-t-il se passer et pourquoi ?

Nous menons une étude sur les difficultés des enfants ayant un trouble du déficit d'attention et hyperactivité (TDAH).

Pourquoi es-tu concerné ?

Tu peux participer à cette étude si tu as entre 10 et 16 ans et si tu as un TDAH. Par contre, tu ne peux pas participer si tu penses que tu ne pourrais pas suivre la procédure.

Qu'est-ce qui t'attend ?

Si tu es d'accord de participer, nous t'inviterons à faire des activités sur l'ordinateur. Ces activités ressemblent à un jeu de mémoire dans lequel tu dois mémoriser des couleurs ou des lettres. Parfois, tu verras une flèche et nous te demanderons de bien faire attention à la couleur pointée par la flèche. Parfois, nous te demanderons de répéter quelques syllabes « ba bi bou » à voix haute en même temps que tu mémorises les lettres, ou de dire si un chiffre est pair ou impair.

Chaque activité dure au maximum trente minutes, et tu devras rester assis devant l'écran de l'ordinateur pendant ce temps. Peut-être que tu te seras un peu ennuyé durant les activités, mais Il n'y a aucun risque en participer de ces activités.

Es-tu obligé de participer?

Non, tu n'es pas obligé de participer. La participation est volontaire : toi et tes parents doivent être d'accord, et tu peux te retirer de l'étude à tout moment sans aucune conséquence négative.

Quel bénéfice retires-tu de ta participation à l'étude ?

Cette étude ne t'apportera aucun bénéfice mais pourra aider la recherche à mieux comprendre les personnes qui ont un trouble du déficit de l'attention et d'hyperactivité.

Vers qui peux-tu te tourner ?

Tu peux toujours parler avec les personnes responsables pour cette étude, elles seront disponibles pour éclaircir tes doutes et répondre à tes questions :

Luísa Superbia Guimarães luisa.superbiaguimaraes@unifr.ch
Téléphone Mobile : 076 816 8672

Université de Fribourg, Département de Psychologie,
Rue P.-A.-de Faucigny, 2, 1700 Fribourg

Prof. Valérie Camos valerie.camos@unifr.ch
Téléphone : 026 300 7675
Université de Fribourg, Département de Psychologie,
Rue P.-A.-de Faucigny, 2, 1700 Fribourg

Annex 3

Medical history questionnaire

UNIVERSITÉ DE FRIBOURG
UNIVERSITÄT FREIBURG

Cahier de questionnaires pour une participation à l'étude :

«ATTENTION AND WORKING MEMORY IN CHILDREN WITH ATTENTIONAL AND HYPERACTIVITY DISORDER"

(La mémoire et l'attention dans le TDAH - titre usuel abrégé)

Madame, Monsieur,

Par la présente, vous recevez un ensemble de questionnaires portant sur les comportements de votre enfant. Remplir ces questionnaires est la première phase de notre étude : c'est une phase dite de sélection d'enfants pouvant y participer.

Nous vous rappelons que le but de cette recherche n'est pas de se substituer à une investigation médicale afin de déterminer la présence ou non d'un diagnostic comme un trouble de l'attention avec/sans hyperactivité et impulsivité (TDAH). Les questionnaires utilisés dans cette étude fournissent des informations utiles sur le plan scientifique, mais ceci sans poser de diagnostics cliniques. La procédure diagnostique est réalisée dans le cadre d'une investigation médicale complète incluant le recueil d'informations et l'observation clinique lors de consultations, les questionnaires apportant des informations complémentaires.

Nous vous demandons de nous renvoyer les questionnaires remplis grâce à l'enveloppe timbrée ci-jointe. Après la vérification des critères d'inclusion et d'exclusion de notre étude, nous vous contacterons par téléphone afin de discuter avec vous du déroulement de la recherche.

Avec nos meilleures salutations,

Mme. Luísa Superbia Guimarães – Investigatrice de l'étude
Prof. Dr. Valérie Camos – Directrice de l'étude

Nom de l'enfant : _____

Date de naissance :

HISTORIQUE MÉDICAL

1. Votre enfant a-t-il été diagnostiqué(e) avec un trouble du déficit de l'attention avec ou sans hyperactivité (TDAH) ?

   ○ Oui

   ○ Non

2. Si oui :

   Date de diagnostic de TDA-H : ⌊_⌋⌊_⌋.⌊_⌋⌊_⌋⌊_⌋.⌊_⌋⌊_⌋⌊_⌋⌊_⌋ (jour.mois.année)

   Le diagnostic de TDAH a été établi par :

   ○ Pédiatre

   ○ Pédopsychiatre

   ○ Neuropédiatre

   ○ Psychiatre

   ○ Médecin généraliste

   ○ Neurologue

   ○ Médecin interniste

   ○ Autre : _____

3. Votre enfant présente-t-il un trouble associé ?

   ○ Oui

   ○ Non

4. Si oui, duquel ? (vous pouvez cocher plusieurs réponses ci-dessous)

   ○ Trouble de la coordination et/ou de la motricité fine

   ○ Trouble du langage

   ○ Dyspraxie

   ○ Trouble du comportement

   ○ Trouble thymique (dépression)

   ○ Trouble anxieux

   ○ Trouble envahissant du développement

   ○ Autre : _____

5. Votre enfant a-t-il reçu un des diagnostics cliniques suivants ?

	OUI	NON
Syndrome d'autisme	☐	☐
Syndrome d'Asperger	☐	☐
Syndrome bipolaire	☐	☐
Psychose	☐	☐
Trouble chronique, tics simples ou multiples, syndrome de Tourette	☐	☐

MÉDICATIONS POUR LE TDAH

Les questions suivantes (numéros 6 à 12) doivent être répondues uniquement si votre enfant a reçu un diagnostic de TDAH.

6. Votre enfant prend-t-il un médicament pour le TDAH ?
   - ○ Oui
   - ○ Non

7. Si oui, depuis _____ (année, mois)

8. Si oui, ce médicament est-t-il :
   - ○ Un psychostimulant (p.ex. Concerta, Elvanse, Equazim, Focalin, Medikinet, Ritaline, etc.)
   - ○ Un non psychostimulant (p.ex. Strattera, Intuniv)
   - ○ Autre : _____

9. Votre enfant a-t-il pris, mais ne prend plus de médicament pour le TDAH ?
   - ○ Oui
   - ○ Non

10. Si oui, votre enfant a pris un médicament pour le TDAH pendant _____ (année, mois)

11. Si oui, ce médicament était-t-il :
   - ○ Un psychostimulant (p.ex. Concerta, Elvanse, Equazim, Focalin, Medikinet, Ritaline, etc.)
   - ○ Un non psychostimulant (p.ex. Strattera, Intuniv)
   - ○ Autre : _____

12. Si oui :
   Pour quelle(s) raison(s) votre enfant ne prend plus un médicament pour le TDAH ? (plusieurs réponses possibles)

- ○ Effets secondaires
- ○ Manque d'efficacité
- ○ Prise trop irrégulière
- ○ Décision parent : si oui, pour quelle(s) raison(s) ?
- ○ Autre : _____

Annex 4

Advertising material

LA MÉMOIRE ET L'ATTENTION DANS LE TDAH

La mémoire de travail est l'habileté cognitive de retenir et manipuler des informations à court terme (de quelques secondes à quelques minutes). Elle est absolument essentielle pour accomplir des tâches comme les calculs mathématiques, la compréhension verbale ou le raisonnement. Soit à l'école (lire, écrire, faire des calculs) ou dans la vie quotidienne (se rappeler d'une liste de courses, le parcours à la maison, ranger ses affaires), la mémoire de travail est impliquée quasiment dans toutes nos activités. Récemment, la recherche scientifique a suggéré que les enfants avec un trouble du Déficit de l'Attention et Hyperactivité (TDAH) présenteraient des différences dans le fonctionnement de la mémoire de travail. Notre projet de recherche financé par le Programme de Bourses d'Excellence de la Confédération Suisse cherche à comprendre quels sont les composants de la mémoire de travail qui fonctionneraient différemment chez les enfants et adolescents présentant un TDAH. L'ensemble de notre projet se déroule également dans le cadre d'un doctorat de l'Université de Fribourg.

RECRUTEMENT DE PARTICIPANTS

- Participation volontaire
- Les enfants et adolescents présentant d'un Trouble du Déficit de l'Attention/Hyperactivité (ou TDAH) qui a été diagnostiqué par un spécialiste.
- Garçons et filles entre 10 et 16 ans.
- L'accord des parents ou des représentants légaux est requis, ainsi que l'aval du médecin traitant
- Les enfants et adolescents ne doivent pas présenter de troubles tels qu'un trouble intellectuel, trouble psychotique, trouble du langage, démence ou tout autre trouble qui empêcheraitde suivre la procédure de l'étude.

Que testons-nous?

Le fonctionnement de la mémoire de travail et ses relations avec l'attention des enfants et des adolescents présentant un Trouble de Déficit de l'Attention/Hyperactivité (TDAH).

Comment se déroule notre recherche?

Étape 1

Les parents / représentants légaux répondent à des questionnaires* sur le comportement de l'enfant ou de l' adolescent

Étape 2

Trois activités seront présentées aux participants, la plupart ressemblent à des jeux sur ordinateur. Chaque activité dure environ trente minutes.

*toutes les données seront traitées de manière confidentielle

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Figure 24. Advertising poster (format A3) for the recruitment of participants. The posters were displayed in public spaces in the city of Fribourg. All the advertising materials were approved by the local ethical authorities in human research.

Figure 25. Advertising flyer, front page (format A5) for the recruitment of participants. The flyers were distributed in health clinics and educational services in different French-speaking cities in Switzerland and handed to families enrolled in the study.

Que testons-nous?

Le fonctionnement de la mémoire de travail et ses relations avec l'attention des enfants et des adolescents présentant un Trouble de Déficit de l'Attention/Hyperactivité (TDAH).

Comment se déroule notre recherche?

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Les parents / représentants légaux répondent à des questionnaires* sur le comportement de l'enfant ou de l'adolescent

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Figure 26. Back page of the advertising flyer (format A5).

Annex 5

Preliminary analysis of the control group

A5.1. – Preliminary analysis of controls in Experiment 1

The following analysis tested if there were differences in performance between participants in the control group without symptoms of inattention and hyperactivity-impulsivity and those whose *T*-scores were inconclusive due to incoherent responses from parents to the questionnaires. We separated participants in two subgroups named "No symptoms" and "Inconclusive" and compared their overall correct response rate in the task by using Bayesian independent samples *T*-tests and we set the alternative hypothesis as $\text{Mean}_{(\text{No symptoms})} \neq \text{Mean}_{(\text{inconclusive})}$.

Table 10 shows the descriptive statistics of the two subgroups of controls in Experiment 1. We found no evidence of a difference between these two subgroups of participants ($BF_{10} = 0.45$, error = 2.575×10^{-5}), therefore we collapsed them into the control group in our main analysis.

Table 10

Descriptive statistics for the correct response rate in the control group in Experiment 1, by subgroups according to the results in the symptoms scales

						95% Credible Interval	
	Subgroup	N	Mean	SD	SE	Lower	Upper
Correct response rate	Inconclusive	11	0.906	0.292	0.009	0.889	0.923
	No symptoms	18	0.880	0.325	0.008	0.864	0.896

Note. SD = standard deviation; SE = standard error of the mean.

A5.2. – Preliminary analysis of controls in Experiment 2

We ran the Bayesian *T*-tests to compare the mean spans and the percentage of letters correctly recalled by the two subgroups of controls in each experimental condition of

Experiment 2. Again, we set the alternative hypotheses as $Mean_{(No\ symptoms)} \neq Mean_{(inconclusive)}$ for each pair of dependent variable. Table 11 shows the descriptive statistics of the two subgroups of controls in each experimental condition of Experiment 2 (fast pace and normal pace) and Table 12 shows the Bayes factors for each pairwise comparison. We found no evidence of differences between the subgroups in all experimental conditions and for the two dependent variables measured (all Bayes factors inferior to 1), therefore we collapsed them into the control group in our main analysis of Experiment 2.

Table 11

Descriptive statistics of the spans and percentage of correct letters of the control group in Experiment 2, by experimental condition and subgroups

						95% credible interval	
Variable (Condition)	Subgroup	N	Mean	SD	SE	Lower	Upper
Span (Slow pace)	Inconclusive	8	4.338	2.026	0.716	2.644	6.031
	No symptoms	11	4.482	1.410	0.425	3.535	5.429
Span (Fast pace)	Inconclusive	8	2.913	2.124	0.751	1.137	4.688
	No symptoms	11	2.509	0.922	0.278	1.889	3.129
% correct (Slow pace)	Inconclusive	8	0.748	0.142	0.050	0.629	0.867
	No symptoms	11	0.740	0.149	0.045	0.640	0.839
% correct (Fast pace)	Inconclusive	8	0.669	0.174	0.062	0.523	0.815
	No symptoms	11	0.569	0.148	0.045	0.470	0.669

Note. SD = standard deviation; SE = standard error of the mean.

Table 12

Bayes factors for each pairwise comparison between subgroups of controls in Experiment 2, by dependent variable and experimental condition

	BF_{10}	Error %
Span (Slow pace)	0.413	4.160×10^{-6}
Span (Fast Pace)	0.456	$5.653^{e} \times 10^{-5}$
% correct (Slow pace)	0.410	2.909×10^{-6}
% correct (Fast pace)	0.753	1.635×10^{-4}

A5.3. – Preliminary analysis of controls in Experiment 3

We ran the same analysis to compare the mean spans and the percentage of letters correctly recalled by the two subgroups of controls in each experimental condition of Experiment 3. We set the alternative hypotheses as $Mean_{(No\ symptoms)} \neq Mean_{(inconclusive)}$ for each pair of dependent variables. Table 13 shows the descriptive statistics of the two subgroups of controls in each experimental condition of Experiment 3 (normal pace and adapted pace) and Table 14 shows the Bayes factors for each pairwise comparison. We found no evidence of differences between the subgroups in all experimental conditions and for the two dependent variables measured (all Bayes factors inferior to 2), therefore we collapsed them into the control group in our main analysis of Experiment 3.

Table 13

Descriptive statistics of the spans and the percentage of correct letters of the control group in Experiment 3, by experimental condition and subgroup

						95% credible interval	
Variable (Condition)	Subgroup	N	Mean	SD	SE	Lower	Upper
Span (Normal pace)	Inconclusive	4	4.333	0.720	0.360	3.188	5.479
	No symptoms	13	4.590	1.047	0.290	3.957	5.222
Span (Adapted pace)	Inconclusive	4	4.083	0.687	0.344	2.990	5.177
	No symptoms	13	4.590	1.355	0.376	3.771	5.409
% correct (Normal pace)	Inconclusive	4	0.662	0.141	0.070	0.438	0.886
	No symptoms	13	0.737	0.085	0.024	0.685	0.788
% correct (Adapted pace)	Inconclusive	4	0.647	0.133	0.067	0.435	0.859
	No symptoms	13	0.735	0.048	0.013	0.706	0.764

Note. SD = standard deviation; SE = standard error of the mean.

Table 14

Bayes factors for each pairwise comparison between No symptoms and Inconclusive participants in Experiment 3, by dependent variable and experimental condition

	BF_{10}	Error %
Span (Normal pace)	0.496	1.926×10^{-5}
Span (Adapted Pace)	0.544	0.001
% correct (Normal pace)	0.795	0.006
% correct (Adapted pace)	1.679	0.002

Annex 6

Supplementary analysis

A6.1. – Supplementary analysis of Experiment 2: Bayesian ANOVA of the spans with age, inattention, hyperactivity as covariates

In this analysis, we included the age and the *T*-scores of inattention and hyperactivity as covariates in the model comparing the mean spans between ADHD and controls in Experiment 2. We ran a Bayesian repeated-measures ANOVA with the group as a between factor, the pace condition as a within factor, and the age, the T-scores of inattention and hyperactivity as covariates in the model. Table 15 shows the Bayes factors for all the models.

Table 15

Bayes factors of each model in the repeated-measures ANOVA comparing the mean spans in Experiment 2, with age, inattention, and hyperactivity as covariates

Model	BF_{10}	Error%
Null model (incl. subject)	1.000	
Pace	22908.878	0.967
Pace + age	15985.803	1.731
Pace + Hyperactivity	12352.466	1.761
Pace + Hyperactivity + age	10813.956	3.866
Pace + groupe	9939.352	1.204
Pace + Inattention	8742.418	2.031
Pace + Inattention + age	7578.733	1.499
Pace + Inattention + Hyperactivity	7552.296	1.957
Pace + Inattention + Hyperactivity + age	6898.843	1.151
Pace + groupe + age	6850.738	2.080
Pace + groupe + Hyperactivity	5091.041	2.168
Pace + groupe + Inattention	4888.037	3.099
Pace + groupe + Hyperactivity + age	4168.445	2.397
Pace + groupe + Inattention + age	3981.343	2.389
Pace + groupe + Inattention + Hyperactivity	3855.710	1.969
Pace + groupe + Pace ✻ groupe	3822.921	3.111
Pace + groupe + Inattention + Hyperactivity + age	3723.190	11.509
Pace + groupe + age + Pace ✻ groupe	2406.808	4.038

Pace + groupe + Hyperactivity + age + Pace ✻ groupe	1986.079	27.709
Pace + groupe + Inattention + Pace ✻ groupe	1854.413	5.753
Pace + groupe + Hyperactivity + Pace ✻ groupe	1829.913	2.190
Pace + groupe + Inattention + age + Pace ✻ groupe	1483.321	5.564
Pace + groupe + Inattention + Hyperactivity + Pace ✻ groupe	1362.769	2.048
Pace + groupe + Inattention + Hyperactivity + age + Pace ✻ groupe	1224.369	5.371
age	0.579	5.998
Hyperactivity	0.432	1.185
groupe	0.362	1.380
Inattention	0.323	1.547
Hyperactivity + age	0.317	0.644
Model	BF_{10}	Error%
Inattention + Hyperactivity	0.241	0.696
Inattention + age	0.234	0.682
groupe + age	0.195	2.020
Inattention + Hyperactivity + age	0.188	0.574
groupe + Inattention	0.158	1.389
groupe + Hyperactivity	0.155	1.615
groupe + Hyperactivity + age	0.112	1.708
groupe + Inattention + age	0.111	2.304
groupe + Inattention + Hyperactivity	0.104	2.348
groupe + Inattention + Hyperactivity + age	0.076	2.152

A6.3. – Supplementary analysis of Experiment 3

A6.3.1. Bayesian ANOVA of the spans with inattention and hyperactivity as covariates

In this analysis, we included the *T*-scores of inattention and hyperactivity as covariates in the model comparing the mean spans between ADHD and controls in Experiment 3. We ran a Bayesian repeated-measures ANOVA with the group as a between factor, the pace condition as a

within factor, and the *T*-scores of inattention and hyperactivity as covariates in the model. Table 16 shows the Bayes factors for all the models.

Table 16

Bayes factors of each model in the repeated-measures ANOVA comparing the mean spans in Experiment 3, with inattention and hyperactivity as covariates

Model	BF_{10}	Error%
Null model (incl. subject)	1.000	
Pace	0.428	1.428
T-Score Hyperacvitiy	0.360	1.688
T-Score Inattention	0.354	1.028
Group	0.352	0.819
T-Score Inattention + T-Score Hyperacvitiy	0.177	1.104
Pace + T-Score Inattention	0.157	5.670
Pace + Group	0.151	2.867
Pace + T-Score Hyperacvitiy	0.149	2.271
Group + T-Score Inattention	0.144	1.122
Group + T-Score Hyperacvitiy	0.142	2.888
Pace + Group + Pace ✻ Group	0.084	1.974
Pace + T-Score Inattention + T-Score Hyperacvitiy	0.079	1.941
Group + T-Score Inattention + T-Score Hyperacvitiy	0.075	1.408
Pace + Group + T-Score Inattention	0.064	2.497
Pace + Group + T-Score Hyperacvitiy	0.059	1.570
Model	BF_{10}	Error %
Pace + Group + T-Score Inattention + Pace ✻ Group	0.034	2.526
Pace + Group + T-Score Hyperacvitiy + Pace ✻ Group	0.033	2.220
Pace + Group + T-Score Inattention + T-Score Hyperacvitiy	0.032	1.711
Pace + Group + T-Score Inattention + T-Score Hyperacvitiy + Pace ✻ Group	0.018	2.032

A6.3.2. Exploratory analysis: correlations between the spans, the CL, and the T-scores of inattention and hyperactivity

We ran a Bayesian correlational analysis to examine if the levels of inattention and hyperactivity were associated with the cognitive load effect (CL) in Experiment 3. We hypothesized that participants with higher levels of inattention would benefit from a decrement in the CL in the adapted pace (because they have slower RTs) whereas participants with higher levels with hyperactivity would suffer from an increase in the CL (because they have faster RTs). This exploratory analysis did not reveal any correlation between the T-scores, the spans, and the CL. Table 17 shows all the Pearson correlations and the respective Bayes factors.

Table 17

Bayes factors and Pearson's correlations between the spans, the CL, and the *T*-scores of inattention and hyperactivity in Experiment 3.

Variables		*T*-Score Inattention	*T*-Score Hyperacvitiy	Span (Normal pace)	Span (Normal pace)	CL (Normal pace)	CL (Adapted pace)
T-Score Inattention	Pearson's *r*	—					
	BF_{10}	—					
T-Score Hyperacvitiy	Pearson's *r*	0.458	—				
	BF_{10}	**4.505**	—				
Span (Normal pace)	Pearson's *r*	-0.074	-0.037	—			
	BF_{10}	**0.247**	**0.235**	—			
Span (Adapted pace)	Pearson's *r*	0.003	0.110	0.259	—		
	BF_{10}	**0.231**	**0.269**	**0.557**	—		
CL (Normal pace)	Pearson's *r*	-0.238	-0.233	-0.005	-0.211	—	
	BF_{10}	**0.481**	**0.467**	**0.231**	**0.410**	—	
CL (Adapted pace)	Pearson's *r*	0.014	0.261	0.078	0.238	0.356	—
	BF_{10}	**0.231**	**0.562**	**0.249**	**0.482**	**1.277**	—

Note. The Bayes factors are in bold for the sake of claritiy.